



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



*Studies from the Institute for
Medical Research, Federated ...*

Kuala Lumpur, Malaya. Institute
for Medical Research

EXCHANGE



STUDIES 8-12

FROM

INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

Vol. 4.

Part 1.

OBSERVATIONS

IN THE

FEDERATED MALAY STATES

ON

BERI-BERI

BY

C. W. DANIELS, M.B. CAMB., M.R.C.S. ENG.

(Late Director Institute for Medical Research, Kuala Lumpur, F.M.S.).

London :

E. G. BERRYMAN & SONS, STEAM PRINTERS,

BLACKHEATH ROAD, S.E.

1906.

Price: Three Shillings and Sixpence.

STUDIES
FROM
INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

Vol. 4.

Part 1.

OBSERVATIONS
IN THE
FEDERATED MALAY STATES
ON
BERI-BERI

BY
C. W. DANIELS, M.B. CAMB., M.R.C.S. ENG.

(Late Director Institute for Medical Research, Kuala Lumpur, F.M.S.).

London :

H. G. BERRYMAN & SONS, STEAM PRINTERS,
BLACKHEATH ROAD, S.E.

1906

Price : Three Shillings and Sixpence.

T. 97

1444

no. 5-12

**BIOLOGY
LIBRARY**

TO VNU
LIBRARY

P R E F A C E .

The publication of this Report is justified by the importance of the subject, the facilities afforded for the study of the conditions under which Beri-Beri develops in Malaya, and the large amount of work in connection with it that has been done in that country.

The facts observed do not support many of the current theories; I have endeavoured to state these fairly, and to ascertain how far they would explain the occurrence of the disease.

So many observers have already investigated the disease, that it does not appear probable that any method but that of elimination will lead to a knowledge of the truth. Without such knowledge attempts at prophylaxis are merely tentative.

I wish to record my obligations to the Government of the Federated Malay States, the Members of the Medical Profession, and the General Public, for their active and cordial assistance to me in these enquiries.

C. W. DANIELS.

March 10th, 1906.

CONTENTS.

	PAGE.
INTRODUCTORY	3
ETIOLOGY—GENERAL CONSIDERATIONS	4
OCCURRENCE OF BERI-BERI IN PRISONS	5
TAIPING	5
BATU GAJAH	9
PAHANG	12
SINGAPORE	13
PENANG	16
KWALA LUMPUR	18
CONCLUSIONS FROM OBSERVATIONS IN THESE PRISONS	39
PLACE INFECTION	41
RELATION OF PRISON EPIDEMICS TO THE GENERAL CONDITION OF THE PEOPLE, AND TO THE GENERAL PREVALENCE OF BERI-BERI	42
MINES	46
PAHANG CORPORATION MINES	49
EARTH, GROUND OR PLACE INFECTION	58
ARSENICAL AND ALCOHOLIC POISONING	60
NITROGEN STARVATION	61
RICE AS A POSSIBLE CAUSE	62
PERIOD OF EXPOSURE NECESSARY FOR THE DEVELOPMENT OF BERI-BERI	63
RICE AS AN OCCASIONAL CAUSE	64
DR. BRADDON'S VIEWS	71
DR. HAVILAND'S VIEWS.. .. .	80
LATENT PERIOD	87
POSSIBLE MODES OF INFECTION	88
CONSIDERATION OF POSSIBLE INTERMEDIATE HOSTS	92
SPECIAL SOURCES OF INFECTION IN PRISONS	96
EARLY SYMPTOMS AND COURSE OF DISEASE	99
CONCLUSIONS	105

BERI-BERI.

OBSERVATIONS IN THE FEDERATED MALAY STATES,

BY

C. W. DANIELS,

Late Director Institute for Medical Research, Kuala Lumpur, F.M.S.

BERI-BERI has been the subject of much study by many observers for over a century. During a portion of this period it has been confused with other diseases, but with the greater number of the observations there is no doubt that the disease considered was that which is now clearly recognised as Beri-Beri. In spite of these observations, which deal with the disease in many countries and under most varied conditions, though the clinical aspects of the disease and its morbid anatomy are well known, and in the main points generally agreed on, the main facts and factors in the causation and spread of the disease are still in dispute.

In the Federated Malay States and in the Straits Settlements the disease is widely distributed, and numerous epidemics have occurred amongst many different classes and amongst the inmates of large Institutions, such as prisons and asylums. The conditions of life in such Institutions are well known, and have been varied in a known manner from time to time. Important information should therefore be available, derived from these Institutions. Further, not only has the disease been the subject of much careful study by the local medical authorities in charge of Institutions, and the result of their observations published, but it has also been studied in certain of these places by experts such as Dr. Hamilton Wright and Dr. Durham, who were given every assistance by the authorities in their investigations.

Under these circumstances, in my opinion, a publication of the main factors in the occurrence of the disease in these public Institutions and amongst certain other bodies of men appears to me to be of considerable importance, and does aid in the elucidation of some of the disputed points in connection with its etiology.

In my inquiries I have limited myself to the last ten years, as it is very difficult to obtain corroborative evidence or accurate information in earlier periods, and even during this period certain points are dependent on the memory and accuracy of the observers. The most important points and the tables are abstracted from the official records made at the time or very shortly afterwards.

A further advantage of an enquiry in such a country as the Federated Malay States is, that the disease is not there a new one, and every Medical Officer has had abundant opportunities for the study of the disease, and is accustomed to be on the look-out for it, so that errors of diagnosis, by the Medical Officers and even by Dressers, are comparatively rare. Occasional doubtful cases may be included in the returns, and no doubt isolated cases of peripheral neuritis from other causes such as alcohol, arsenic, diphtheria, &c., if they occur, would probably be mistaken by the Assistant Surgeons, and

possibly by some of the Medical Officers, for Beri-Beri. Such or other mistakes in the prisons are, I believe, from my own observation, extremely rare. Their occasional occurrence should be remembered as a warning against laying any stress on the occurrence of *isolated* cases diagnosed as Beri-Beri. It is not, in my opinion, justifiable to rely so implicitly on the diagnosis as to attach any value to the reported occurrence of a single isolated case of the disease.

ETIOLOGY.

The great difficulty that has arisen in the study of the etiology of the disease is that the clinical symptoms are those of a wide-spread lesion of the nervous system, which is certainly secondary; and that the primary lesion is yet unknown, none of the suggested ones being universally accepted. The symptoms associated with the primary lesion, if one occurs in man, are indefinite. If the primary cause of the nerve lesions is formed outside the human body, its nature is unknown with certainty.

The secondary nature of the nerve lesion is a point emphasized by Dr. Hamilton Wright. Much confusion would be avoided if the following distinction was always kept in mind, namely, that the lesions, which form the group of symptoms known clinically as Beri-Beri, are a secondary result.

This seems to me to be clearly established both by the consideration of the observed lesions and also by analogy with the other forms of neuritis with known etiology.

The main hypotheses which have been suggested, and which recognise the secondary nature of the lesion, can be grouped into two.

- (1) That the disease known as Beri-Beri is secondary to a lesion or disease occurring in the same man.
- (2) That Beri-Beri is secondary to the absorption by man of a poison generated or existing outside the human body.

The first hypothesis includes that of Dr. Hamilton Wright, who drew renewed attention to a condition of the stomach and duodenum frequently present in cases of Beri-Beri. This condition he considers to be a gastro-duodenitis, and to play a similar part in the causation of the neuritis that the throat lesion does in post diphtheritic peripheral neuritis. In sections of the stomach and duodenum, in this condition, he has demonstrated the presence of a large bacillus.

Dr. H. P. Durham considered the throat as a possible site for a primary lesion, and in congested areas, which occur in this situation, he demonstrated another organism with peculiar cultural characters. Some of the older observers found various organisms in the blood, but all recent observations conducted with full precautions have failed to find such organisms.

The primary source, if in man, induces only slightly marked symptoms, as the prodromata are ill defined and indefinite in a large proportion of cases.

Some of the analogies point to an infection with parasites of the protozoal nature; but such parasites have not been found even in the early stages of the developed secondary disease—neuritis—and if they exist before the nerves are affected, produce no definite symptoms, not even pyrexia.

According to any of these theories, the disease would necessarily be one conveyed from man to man, either directly or indirectly, *via* air, water, or food, or by an intermediate host.

The second hypothesis includes all theories in which the cause is ascribed to infected ground in which this agent develops. By absorption or inhalation it is supposed to be taken into the human system, and there exercise its destructive functions on the nerves.

It also includes all theories of poisoning by various articles of food, such as rice, fish, &c., either when sound or diseased.

It further includes poisoning by such substances as are known to induce peripheral neuritis, whether inorganic as in the case of arsenious oxide, or organic such as alcohol, or other substances which produce similar effects on the nerves.

The following histories of the conditions under which Beri-Beri has been prevalent include those which may possibly, according to any of the modifications of these hypotheses, be causally related to the occurrence of Beri-Beri. The plan adopted is to give the history of the occurrence of Beri-Beri in these Institutions, and, where the incidence has varied greatly, to divide into periods according to such variations, and to relate such circumstances as bear on any of the suggested theories.

I take first the prisons.

Most of the literature already published deals with the outbreaks in Singapore and Kwala Lumpur, but outbreaks have occurred in all, even in the last ten years or so.

PRISON BERI-BERI IN MALAYA.

In the Taiping Gaol since 1883, with about the same number of prisoners as Kwala Lumpur, there have been no big epidemics. The average prison strength in 1894 was 414, and is now between 500 and 600; and a certain proportion of prisoners are admitted direct from Batu Gajah Prison, supplied from a considerable district, the Kinta Valley. All the short-sentence prisoners, with sentences of under three months, are detained at that gaol, and even those sentenced to longer terms are kept there whilst awaiting trial, sometimes for three months, and are transferred subsequently to receiving their sentences after a variable period, usually less than one month. A larger number of cases occur at Batu Gajah than at Taiping, and, as will be shown, the "endemic index" there is exceptionally high.

A table of the occurrence of cases in Taiping Gaol, excluding as far as possible relapses, but making no distinction as to whether before imprisonment the patients had suffered from Beri-Beri, is here given. It is abstracted from the Prison Hospital register, kindly supplied to me by Dr. M. J. Wright, State Surgeon, Perak.

The noteworthy point, in contrast with Kwala Lumpur, is the frequency of prolonged intervals during which no cases of Beri-Beri occurred. Only one of these intervals corresponds with an interval at Kwala Lumpur, and, even in this instance, the correspondence is not close, as the interval in Kwala Lumpur was from February to November, 1903, whilst at Taiping it was from May, 1903, to January, 1904. The Taiping Gaol has the advantage of being built in separate blocks, and allows much freer access of air to the different buildings. The ventilation of the cells was greatly superior to that in Kwala Lumpur prison, before the alterations were made in May, 1903. Even now, though there is not the same provision for ground ventilation as in Kwala Lumpur, the actual ventilating area is greater: eight square feet as against

six square feet outside ventilation. The rice supplied is Rangoon rice, and it has been supplied during the whole period under review. The changes in the dietary have been slight.

TABLE A—Taiping Gaol.

	1893.	1894.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.	1903.	1904.	1905.
Jan.	1	2	6	...	6
Feb.	2	7	1	5	4	1	2
Mar.	1	...	2	7	...	1	2	10	4
April	2	4	...	4	1	2	...
May	2	1	...	1	4	...	3	...
June	4	1	...	3	...
July	2	1	3	...	5	...
Aug.	3	1
Sept.	3	3	...	1	...
Oct.	2	1	2	8	...	1	...
Nov.	5	3	7	4	...	1	...
Dec. ...	1	1	3	3	1	9	...	6	...
TOTAL	1	4	...	16	11	23	16	42	13	33	12

In 1890, 1891 and 1892 there were no cases.

The rice is steamed under slight pressure, and in the morning Kangi has always been served hot. The work, discipline, and general administration in Taiping is, and has been, similar to that in Kwala Lumpur. Though the mortality is low, dysentery and diarrhoea have been much more prevalent than in Kwala Lumpur.

The prisoners come from Taiping and the surrounding districts, where Beri-Beri is only moderately common, and from Batu Gajah Gaol, with a high endemic index, so that it is important to determine in what proportion of these cases the disease is really acquired in this gaol. The medical examination has not been directed to the same extent to the determination of previous attacks of Beri-Beri as in Kwala Lumpur, so that it is impossible now to ascertain if any of those cases attacked had previous attacks outside.

Some of the cases may be relapses. Some certainly were infected outside or in the Batu Gajah Gaol, as is shown by the short interval that elapsed between admission to the prison and development of the disease.

TABLE B—Taiping Gaol.

No admissions, no cases, no relapses from Mar. 22nd, 1894, to Mar., 1896.

	Less than 2 weeks.	Less than 1 month.	Less than 2 months.	Over 2 months.	Relapses.
1896.					
January
February
March ...	1	1
April ...	1	1	...
May	2	...
June ...	1	1	...	2	1
July	2	...
August	1	...	2	...

No admissions, no cases, no relapses from Sept., 1897, to Feb., 1898.

TABLE B—TAIPING GAOL—*continued.*

	Less than 2 weeks.	Less than 1 month.	Less than 2 months.	Over 2 months.	Relapses.
1898.					
March	1
July	1
August	1
These three relapses were all in the same patient.					
1899.					
June	1	...
No admissions, no cases, no relapses from Sept., 1898, to May, 1899.					
No admissions, no cases, no relapses from July to Sept., 1899.					
October	3 M.G.	1 M.G.
November	3 M.G.	1 M.G.
December	2 M.G.	2 M.G.
1900.					
January	2 M.G.	2 M.G.
February	1 M.G.	1 M.G.
March	7 M.G.	4 M.G.
April	4 M.G.	6 M.G.
May	1 M.G.
No admissions, no cases, no relapses for June and July.					
August ...	1
September
October	1
November	2 M.G.	1
December	3 M.G.	...
1901.					
January
February	1
March	1
April
May	1
June
July	1 L.G.	...
August
September ...	1	...	1
October	1
November	1	6 { 3 M.G. 3 L.G. }	2
December
1902.					
January
February ...	2	1	2
March ...	5	1 L.G.	...
April ...	1	1	3
May ...	2	1 M.G.	...
June	1
July ...	1	2 M.G.	2
August	1
September ...	1	2
October ...	1	6 { 5 M.G. 1 L.G. }	3
November	4 { 3 M.G. 1 L.G. }	1
December	7 { 6 M.G. 1 L.G. }	3
1903.					
January	5 M.G.	1
February	4 M.G.	...
March	2 M.G.	1
April	1 M.G.	...

No admissions, no cases, no relapses from May, 1903, to Jan., 1904.

TABLE B—TAIPING GAOL—*continued.*

	Less than 2 weeks.	Less than 1 month.	Less than 2 months.	Over 2 months.	Relapses.
1904.					
February ...	1
March ...	3	2	...	6	...
April ...	1	2	1
May ...	1	1	...	1	...
June ...	2	1
July ...	2	1	...	2	...
August
September ...	1
October	1	...
November	1	...
December ...	2	1	...	3	...

There were also seven persons admitted to the gaol between April and December, 1904, suffering from Beri-Beri.

In 1903, all the cases occurred after more than six months' imprisonment.

No further cases, no relapses and no admissions with the disease during the next ten months from April 6th, 1903, to February 25th, 1904, when a prisoner who had been 13 days in the prison developed the disease. He is stated to have been ill for three days before admission to hospital.

TAIPING PRISON.

Preceding the first small outbreak in 1896, three cases occurred in prisoners who had been respectively 20 days, 4 days, and 10 days in gaol, and during the outbreak two other prisoners developed the disease in less than two weeks and two in less than one month.

No other cases developed for 19 months, but in March, 1898, one patient, who had previously had the disease, had an attack, and relapsed in July and August.

For a further period of nine months there were no cases, but a solitary case in a prisoner who had been seven years in gaol with no previous history occurred in June.

Three months later there was a relapse case, and in the next eight months there were 17 relapses and 24 new cases, all Middle or Upper Grade prisoners, and all more than one year, and most of them more than two years, in gaol.

From May, 1899, to September, 1900, there was one relapse, and one patient admitted with the disease, and in October and November there were in each month a relapsed case, and in November and December five persons, all in the Middle Grade, were attacked. There was a further interval of six months broken by three relapses, and then a prisoner six months in gaol was attacked. Four months later there were two relapses and six fresh cases.

In February, 1902, there were three persons admitted who had Beri-Beri in less than one month, and two relapsed cases, and during the rest of the year 14 cases were admitted and 15 relapses occurred, whilst 21 cases originated in the gaol, and the outbreak only gradually died out in the next four months, during which 13 cases occurred.

Whilst the solitary case in June, 1899, is difficult to explain, as there had been so long an interval between that and any previous case, either admission or relapse, the general run of cases is strongly in support of the spread of the disease either from relapsed cases or cases admitted with the disease.

The outbreak in 1899, when all the cases, both relapses and fresh attacks, occurred amongst the prisoners who had served more than one year, and when none occurred amongst those who had served less than 13 months, is decidedly significant of infection by relapsed cases of other prisoners in the same grades, and is absolutely opposed to any theory of causation of the disease by the class or quality of rice used. A reference to the diet table for this period will show that the disease occurred amongst those whose diet was supplemented by other articles, and who were not taking the largest amount of rice. It has also been shown that the conditions necessary to produce Beri-Beri, either amongst Sinkehs or in Kwala Lumpur Gaol, take only some three or four months to have their full effect. If the food or any article of the food produced the effect, it must have taken a minimum of 13 months to produce its effect in this instance. The relative number of prisoners under and over one year incarceration cannot be determined, but those who have been in gaol at any one time for less than a year, always exceeds those who have been over a year.

It will be noted that in the first quarter of 1903, the disease was endemic, and affected only prisoners who had been more than six months in gaol, and that the outbreak was subsiding steadily during this period, and that no fresh cases from outside were admitted.

In 1904, following and associated with the admission of prisoners with the disease, there was a recrudescence, and cases continued to occur amongst the prisoners all through the year, but only a small proportion were affected. These prisoners included men in all grades.

BATU GAJAH GAOL.

The Batu Gajah Gaol is peculiar, in that only short-sentence prisoners are kept there, and prisoners from the district who are awaiting trial. The district is a large one, and Dr. Fox (the Senior District Surgeon), estimates the population at 130,000. Beri-Beri occurs in most of the mines; and from the Kinta District in 1904, 1,433 cases of Beri-Beri were admitted to various hospitals, a rate of 11.02 per 1,000, half the rate for the Rawang Mining District. The admissions to the Gaol Hospital for Beri-Beri, are given in the subjoined table:—

Occurrence of Beri-Beri in the Batu Gajah Prison.

TABLE A.

	1898.	1899.	1900.	1901.	1902.	1903.	1904.	1905.
January	1	2	13	4	...	2	2
February	8	7	12	14	3	1	2
March	9	4	10	4	...	2	1
April	5	11	6	7	...	1	1
May	6	5	18	6	3	6	...
June	13	15	13	4	4	17	...
July	5	8	8	23	14	...	13	...
August	3	6	10	13	3	...	5	...
September	5	3	6	5	7	2	8	...
October	16	4	13	10	3	2	5	...
November	19	15	10	11	4	1	4	...
December	3	11	16	15	3	8	27	...
TOTAL	51	89	107	149	73	23	91	6

They are seen to be much in excess of those at Taiping, and with the exception that the rate was low in 1903 in both, the two returns show no

correspondence. An analysis of the figures, however, shows that the high rate at Batu Gajah is mainly due to the number of persons admitted with Beri-Beri.

TABLE B—Batu Gajah.

	Less than 1 week.	Less than 2 weeks.	Less than 1 month.	Less than 2 months.	Over 2 months.	Total.
1901.						
July ...	8	...	5	7	3	23
August ...	5	2	2	1	3	13
September ...	4	1	...	5
October ...	2	1	5	1	1	10
November ...	1	3	5	2	...	11
December ...	6	1	...	8	...	15
TOTAL ...	26	7	17	20	7	77
1902.						
January ...	2	1	...	1	...	4
February ...	6	3	1	...	4	14
March ...	2	2	...	4
April ...	5	...	1	1	...	7
May ...	1	2	3	6
June	2	2	4
July ...	10	2	2	14
August	1	1	2
September ...	6	1	7
October	2	2	4
November ...	2	1	1	4
December ...	2	1	3
TOTAL ...	36	14	10	6	7	73
1903.						
January
February ...	2	2
March
April
May ...	1	1	...	2	...	4
June ...	2	1	...	1	...	4
July
August
September	1	1	...	2
October	2	2
November	1	1
December ...	3	...	1	3	1	8
TOTAL ...	8	3	4	7	1	23
1904.						
January ...	2	2
February	1	1
March ...	2	2
April ...	1	1
May ...	4	...	2	6
June ...	1	4	6	5	1	17
July ...	1	1	5	3	3	13
August ...	1	...	1	3	...	5
September	2	2	...	3	7
October ...	1	...	1	1	2	5
November	1	1	1	1	4
December ...	2	9	7	6	2	26
TOTAL ...	15	17	25	19	13	89

In these $3\frac{1}{2}$ years, 65 persons developed Beri-Beri in a week or less, and of these, 44 were imprisoned for "vagrancy." Thirty-five more developed Beri-Beri after more than a week, but in two weeks or less; eight of these were vagrants.

One hundred and twenty-seven developed Beri-Beri in a month or less, out of the total of 199 in these $3\frac{1}{2}$ years, making nearly two-thirds of the total cases. In these, the infection had probably been acquired before admission.

Of the remaining 72, 40 developed the disease within the next month. Some, at least, of these may have been infected before admission.

The remaining 27, an average of 7.7 per year, were almost certainly infected in the gaol. Some may have been relapses in persons who had previously suffered from Beri-Beri. It must be remembered that, as prisoners sentenced to less than three months only are retained in this gaol, the number of inmates over two months is small.

It is clear that cases do originate in this gaol, as some show themselves over two months after admission. With so many infected short-sentenced prisoners, it is to be feared that, in this gaol, many persons become infected, and are discharged or transferred before the development of the disease, and so it becomes a kind of incubating house and distributing centre for the disease.

Out of the cases occurring in Taiping Gaol since June, 1902, 23 S.S. and L.G., 32 M.G., and 16 U.G., were in persons transferred from Batu Gajah Gaol, making a total of 71 out of 108 cases, though the relative strength of the Batu Gajah contingent is only about one-fifth of the whole.

**Prisoners Transferred from Batu Gajah to Taiping Prison
who developed Beri-Beri whilst in Taiping Prison.**

		S.S.	L.G.	M.G.	U.G.
1902	...	3	10	16	7
1903	...	—	—	10	3
1904	...	7	2	4	5
1905	..	—	1	2	1

From Ipoh to Taiping Prison.

		S.S.	L.G.	M.G.	U.G.
1902	...	—	—	1	—
1903	...	—	—	—	—
1904	...	1	1	2	—
1905	...	1	1	1	—

From Taiping.

		S.S.	L.G.	M.G.	U.G.
1902	...	—	—	1	—
1903	...	—	—	—	—
1904	..	2	—	—	—
1905	...	2	—	1	—

Beri-Beri in Prisoners Transferred to Taiping Prison.

From—	1902.	1903.	1904.	1905 (1st quarter).
Batu Gajah Gaol	34	13	18	4
Taiping	9	—	2	3
Ipoh	1	—	4	3
Krian	—	2	2	—
K. Kangsar	2	—	2	—
Tapah	—	—	—	1
T. Anson	—	—	—	2

The cells in Batu Gajah Gaol are similar, in most respects, to those at Taiping, but there are only 156 of them. The other prisoners are confined in well-ventilated association wards. Until recently, all the work was light but now stone-breaking has been introduced.

The rice supplied is No. 2 "Siam," grown in Province Wellesley and prepared in Penang. It is a more expensive and better rice than is supplied to Taiping or Kwala Lumpur.

In both Taiping and Batu Gajah no mats are supplied, only blankets and wooden pillows. The blankets are not washed, but, after use, are sunned, stored, and issued to fresh convicts.

PAHANG PRISONS.

The prisons at Pahang are smaller, and differ in the following important points :—

1. The buildings are wood, with wooden floors, and atap roofs.
2. Most of the prisoners are in association wards, and not in separate cells.
3. Much of the work (water carrying, &c.), is extra-mural.

At Pekan, the following cases occurred :—

1897.	1898.	1899.	1900.	1901.	1902.	1903.	1904.
8	—	2	5	3	4	—	1

The solitary case in 1904 had previously had Beri-Beri, and probably was a relapse.

At this prison the Chinese do not form the great bulk of the prisoners, and the incidence was therefore not specially amongst the Chinese; 12 of the cases were Malays, 7 Chinese, and 4 Bengali or Tamils.

Occurrence in months :—January, 5; February, 5; March, 2; April, 2; May, 1; June, 1; July, 2; August, September and October, Nil; November and December, 4.

In December, January and February 14 out of 23 cases occurred. The average strength of the prison is only 20, and nearly all are short-sentence prisoners. Siamese rice is used exclusively.

Beri-Beri In Kwala Lipis Gaol.

	1897.	1898.	1899.	1900.	1901.	1902.	1903.	1904.
January	1	1
February	...	1	1
March	...	1	2	...
April	...	2	2	3
May	...	1	1	1	...	1	...	1
June	...	2	...	4	1	...	1	...
July
August	...	1
September	1	...
October	...	2
November	...	2
December	...	1	1
TOTAL	13	10	4	8	2	1	5	2

Of the cases in 1901 and subsequent years, 10 in all, 3 were admitted with or developed the disease in less than a week. These cases occurred in December, 1901, January, 1903, and April, 1904. Two cases occurred in persons who had been over 1 year, 1 in a person over 6 months, and 2 in persons over 3 months in the prison, so that these, half the total cases, had probably originated in the gaol.

The rice used is "Rangoon," and the average prison strength 77 to 96. Much of the work is extra-mural.

In the gaols in the Straits Settlements, Beri-Beri has frequently occurred, and in some years there have been severe epidemics. In the subjoined table the occurrence in the Singapore Prison is given; relapses and admissions with the disease, are included in this table. The prison is older than those in the Federated Malay States; the buildings are crowded together, and the ventilation of the cells is worse than in the other prisons. Improvements have recently been made.

SINGAPORE GAOL.

Statement of Beri-Beri Admissions and Deaths, Prison Hospital Singapore—1894 to June, 1905.

MONTHS.	1894.		1895.		1896.		1897.		1898.		1899.		1900.		1901.		1902.		1903.		1904.		1905.	
	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.	A.	D.
Jan.									1	22		5	15	1	22	1	22	2	30	2	3			
Feb.				1				1	25		3	10		8	1	6		13		21				
Mar.				2					25		1	4		2		6		20		1				
April								1	23		8	17		8		3		16						
May								2	5		14	2	25	1	18		1		28	2	1			
June									7	2	32	2	17	3	79	3	3		11	1	7			
July								1	7		36	2	23	2	86		1		29	1				
Aug.								2	19		8	1	12		27				34	2				
Sept.								6	3	†	32	1	10		36	3	2		45	7				
Oct.								10			5	†	24	2	19	1	62	1	7		26	1		
Nov.							2	78	1		8		44	7	41	1	35	1	53	6	10			
Dec.							1	22		16		17	11	26		32	1	65	8	4	1			
TOTAL				3		3		124	1	165	4	224	28	219	9	415	11	169	16	266	17			

* 20 old cases returned from Malacca.

† Cirrhosis of Liver and Tubercle of Peritoneum.

‡ Dysentery.

It will be seen that, in 1894, there were no cases, and that in 1895, 1896 and 1897, when no less than 911 cases occurred in Kwala Lumpur, and 16 in Taiping Gaol, only 6 cases occurred in Singapore (3 in February and March, 1896, and 3 in November and December, 1897), though the average strength was about twice as great in Singapore. The fall in the price of tin must have affected, to some extent, the condition of the poorer classes in Singapore, but would not have led to an influx of destitute mining coolies as in Kwala Lumpur.

In 1898, in the last six months, the cases became numerous, and were so all through 1899, but considering the difference in numbers, it was not so great as in Kwala Lumpur Prison.

From 1900 to January, 1903, there was a general correspondence to the returns from Kwala Lumpur; in both 1900 and 1901 there was a decided

increase in the number of cases, and in 1902 nearly double the number in 1901, followed by a very great decrease in January, 1903. Here the correspondence ceases, as there was a severe outbreak at the end of 1903, continued throughout 1904, till the end of the year.

The history of the outbreak in November, 1903, is of interest, as the decline at the end of January corresponded to a decline at the Lunatic Asylum in the Kwala Lumpur Gaol, and to some extent, at the Taiping Prison, and on Christmas Island.

In the Institutions in the Malay States, the cessation of admissions to gaol of prisoners in the early stage of Beri-Beri was associated with this decrease, and this cessation appeared to be the result of a diminution in the amount of crime amongst the miners consequent on the establishment of more amicable arrangements between the mine-owners and their employés.

Singapore is only to a minor extent dependent on the prosperity of the Federated Malay States or tin mining. The most important rôle is that of a large distributing centre for the Far East.

From Singapore, Beri-Beri was introduced into Christmas Island, and according to Durham's Report, further introductions have taken place. It is therefore no matter of surprise that a diminution in Beri-Beri in Singapore evidenced by a diminution in the cases in the prison and in the Lunatic Asylum, should be associated with a corresponding diminution in Christmas Island, a place which receives all supplies, and also Beri-Beri *via* and from Singapore.

There is no necessity to assume that the Rangoon rice retained for consumption in Singapore and exported to Christmas Island, contained a poison producing Beri-Beri, whilst the Rangoon rice supplied to Kwala Lumpur and Taiping and Mombasa, and elsewhere, from Rangoon did not contain such a poison.

The rice has been frequently changed, but the changes seem to have been between "Siam" and "Rangoon" rices, both rices that are husked without previous boiling or steaming.

From January, 1903, to October, 1904, Rangoon rice alone was used according to Dr. Leask, the Medical Officer attending the gaol.

In October, 1904, Rangoon, except for 2ozs. used for Kangi, was discontinued, and rice soaked in water and rapidly steamed before husking was used for all other purposes. This so-called "Bengal" rice was grown in Kedah or Province Wellesley, and prepared in Penang. There was a striking diminution in the number of cases of Beri-Beri, and early in 1905, this "Bengal" rice was also used, after washing, for Kangi. The Beri-Beri rate for the first six months of 1905 was even lower than from February to October, 1903, when Rangoon rice only was used. Extensive and much needed improvements in the sanitation of the gaol have been carried out during 1904, these include improved ventilation of the cells, better protection from rain and wind, and improved water supply, and removal of certain risks of contamination of food by faecal matter.

The detailed history of the outbreak in 1903 was compiled from the official records and my own observations for a part of the time, but for much of the information I am indebted to Dr. McDowell, P.C.M.O., Dr. Leask, and Mr. Elcum, the Superintendent of the Prison.

There had been no fresh cases and no admissions with the disease for more than three months before the commencement of the outbreak in September. The first case was diagnosed in a prisoner who had been less than five weeks in the prison, and had therefore possibly acquired the disease outside. In October there were two cases, both short-sentence prisoners, but one of these must have acquired the disease in the prison, as he had been an inmate for more than three months. In November, there were 32 cases, including six who had been over a year in the gaol, and in December, 41, five of whom had been prisoners for over a year. The cases during this period were mainly amongst short-sentence prisoners, 64, and amongst long-sentence prisoners, 13, during the first year of their imprisonment, when they are engaged in the same class of work, and are under nearly the same conditions as the short-sentence prisoners. These two classes contributed therefore 77 cases out of 92. Of the prisoners over a year in gaol, 13 only were attacked, and of the Upper Grade prisoners, those who had been one year plus half their remaining sentence in prison, two were attacked, both of whom had just been transferred from the Middle Grade, so that during the first five months of the outbreak this class escaped.

The average strength of the prisoners under one year was 384, and of the middle grade 284, so that the proportion of those in the first year who were attacked was quite out of proportion to the relative numbers.

These figures do not include relapses of persons admitted with the disease, only those who doubtfully or certainly had acquired the disease in the prison. The official returns appended, give the total number of admissions to hospital for the disease in any form.

I had no further opportunity of studying the progress of the epidemic, but the disease continued to prevail throughout 1904 with fluctuations, but diminished in the first half of 1905. During this period important sanitary reforms alluded to were in progress. Till these are complete it will be difficult to exclude factors such as faecal contamination of food, imperfect ventilation, &c., from a share in the propagation of the disease.

The Penang Prison has been singularly free from Beri-Beri. From 1893—1904 both inclusive, there were only 31 cases, and of these eight developed the disease in two weeks or less after conviction, three more under one month and another three whilst awaiting trial, so that probably 17 only acquired the disease in the gaol in 12 years, and as only eight of them had been over three months in gaol, these alone can be considered as having certainly acquired the disease in gaol, and some of these may have been relapses.

Of the cases in this period, the two in 1893 were admitted to the hospital on the day of conviction and two days afterwards, so that no cases were acquired in the gaol in that year.

The two cases in April, 1895, are stated to have been acquired outside, but as one was only admitted to hospital after three months' incarceration, this may be doubtful.

In 1896, the cases in January and February are stated to have acquired the disease outside, and those in July, August and September were respectively in prisoners awaiting trial, in one three days after conviction, and in another nine days after conviction.

The solitary case in 1899 developed two days after conviction. In brief, no cases acquired the disease in gaol in 1893; 1894, one only, or two in 1895; possibly three in 1896; possibly three each in 1897 and 1898; and none in 1899. In most years, cases were admitted with the disease, but

isolated early, and in this connection there is a difference in the custom in this gaol, as prisoners are kept in the hospital under observation for one or more days before they are considered to be admitted to the hospital, in order that a sure diagnosis may be arrived at. Cases therefore returned as "admitted" to hospital one or two days after conviction, will usually have been sent to the hospital on admission to the gaol under observation, but are only returned as admitted to the hospital when the diagnosis is certain. Such cases therefore cannot infect any prisoners but those in the hospital.

PENANG GAOL.

Penang, though like Singapore to some extent in being a Sea-port, differs in many respects. It is more of an agricultural centre, and the easy communication with the coast districts of the Malay Peninsula brings it into close relation with those agricultural districts. Comparatively few of the mining population in ordinary years migrate to Penang, but in years such as 1896 and 1897, when the mining industry was at so low an ebb that the miners were forced to undertake other forms of work, a certain portion of the labour no doubt was diverted to Penang.

Beri-Beri is both absolutely and relatively less prevalent in Penang than in Singapore. In round numbers, the population of Singapore is some 180,000, and of Penang 75,000.

The deaths from Beri-Beri have been in the last five years as follows:—

	1900.	1901.	1902.	1903.	1904.	TOTALS.
Singapore ...	1,345	1,399	1,091	1,340	1,621	6,796
Penang ...	461	407	313	155	307	1,637

Represented per 10,000 of the population, the figures are:—

	1900.	1901.	1902.	1903.	1904.
Singapore ..	74.7	77.7	60.6	74.4	90.
Penang ...	61.4	53.4	41.7	20.6	40.9

The number of cases admitted to the Pauper Hospital varies, but the variations show little correspondence with those in the Malay Peninsula. In the years 1896 and 1897, when the mining industry was depressed, the number of cases was very large, and diminished with the increased prosperity in 1898, and there was also a decrease in 1903, as in so many places in Malaya.

PAUPER HOSPITAL, PENANG.

	1896.	1897.	1898.	1899.	1900.	1901.	1902.	1903.	1904.
Cases ...	558	645	235	413	355	424	382	234	455
Deaths ...	83	92	15	65	65	134	121	69	136

Occurrence of Beri-Beri in Penang Gaol.

	1893.	1894.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.	1903.	1904.	1905.
January	1	...	1
February	1	1	1	...	1
March	1	1	1
April	2	1	...	1
May	1	1
June	1	18
July	1	17
August	1	...	1	2
September ...	1	1	2
October	1	1	1	1	1	...	1
November	1
December ...	1	1
TOTAL ...	2	...	3	9	3	3	1	3	2	2	4	...	37

In 1893 both cases were in Hospital within two days of conviction.

All the cases in 1895 are said to have acquired the disease before admission to prison.

In 1896, the cases in January and February acquired the disease before admission. The cases in July, August, and September, were in prisoners awaiting trial, three days after conviction, and nine days after conviction respectively.

In 1899, the solitary case developed two days after conviction.

In this gaol, the only outbreak that has occurred was in 1905, for the number of cases that developed in other years was too small for their occurrence to be considered as outbreaks, and some of them, at least, may have been relapses in persons who had acquired the disease before admission to the gaol.

In 1905, there were no cases, either admitted with Beri-Beri or developing it, in the gaol in the first quarter.

On April 6th, one person was sent to hospital on the day of sentence, under observation, and 14 days later was definitely diagnosed as Beri-Beri. No opportunity therefore was given for this case to spread the infection.

On May 16th, another prisoner was passed as fit for hard labour, and at first worked well, but 10 days after admission was admitted to hospital as Beri-Beri, and the case was an acute one, terminating fatally in 14 days. On admission to hospital, he stated that he had been feeling unwell for five days. This prisoner had been confined in the cells, not in the association ward, and the next five cases, all that developed up to June 15th, were in prisoners also confined in cells. Out of the total of 26 who developed the disease between June 17th and July 29th, only seven were confined in the block in which the Civil Prison and the overflow ward from the Criminal Prison are situated; of these, five had been transferred from cells in the Criminal Prison to the Civil Prison, three weeks or less before they were admitted into hospital for Beri-Beri.

A single case, a relapse, occurred in an old woman in the Female Ward during the same period, but she had been suffering from Beri-Beri in a sub-acute form, for a prolonged period. This case, therefore, did not really form any part of the outbreak under consideration.

The occurrence of cases after the case in May, in the patient who was in the Criminal Prison from May 16th to 26th, during which he developed the disease, were as follows, in weeks :—

Week ending—June 1st.	June 8th.	June 15th.	June 22nd.	June 29th.
0	2	3	8	1
Week ending—July 5th.	July 12th.	July 19th.	July 28th.	July 31st.
1	6	4	4	2

One of the cases in the week ending July 28th developed the disease less than one month after admission to the gaol, so he may have contracted the disease outside; with that exception, all the other cases were prisoners who had been two months or more in gaol, and, therefore, in all probability, had contracted the disease in prison.

It will be noted that, assuming the infection was derived from the case in May, the interval between the last possibility of infection, May 25th, and

[illegible]

33.
 34.
 35. E
 36.

— 17 —

..

•

13

11

1. 2. 3. 4. 5. 6. 7. 8. 9. 10. 11. 12. 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100. 101. 102. 103. 104. 105. 106. 107. 108. 109. 110. 111. 112. 113. 114. 115. 116. 117. 118. 119. 120. 121. 122. 123. 124. 125. 126. 127. 128. 129. 130. 131. 132. 133. 134. 135. 136. 137. 138. 139. 140. 141. 142. 143. 144. 145. 146. 147. 148. 149. 150. 151. 152. 153. 154. 155. 156. 157. 158. 159. 160. 161. 162. 163. 164. 165. 166. 167. 168. 169. 170. 171. 172. 173. 174. 175. 176. 177. 178. 179. 180. 181. 182. 183. 184. 185. 186. 187. 188. 189. 190. 191. 192. 193. 194. 195. 196. 197. 198. 199. 200. 201. 202. 203. 204. 205. 206. 207. 208. 209. 210. 211. 212. 213. 214. 215. 216. 217. 218. 219. 220. 221. 222. 223. 224. 225. 226. 227. 228. 229. 230. 231. 232. 233. 234. 235. 236. 237. 238. 239. 240. 241. 242. 243. 244. 245. 246. 247. 248. 249. 250. 251. 252. 253. 254. 255. 256. 257. 258. 259. 260. 261. 262. 263. 264. 265. 266. 267. 268. 269. 270. 271. 272. 273. 274. 275. 276. 277. 278. 279. 280. 281. 282. 283. 284. 285. 286. 287. 288. 289. 290. 291. 292. 293. 294. 295. 296. 297. 298. 299. 300. 301. 302. 303. 304. 305. 306. 307. 308. 309. 310. 311. 312. 313. 314. 315. 316. 317. 318. 319. 320. 321. 322. 323. 324. 325. 326. 327. 328. 329. 330. 331. 332. 333. 334. 335. 336. 337. 338. 339. 340. 341. 342. 343. 344. 345. 346. 347. 348. 349. 350. 351. 352. 353. 354. 355. 356. 357. 358. 359. 360. 361. 362. 363. 364. 365. 366. 367. 368. 369. 370. 371. 372. 373. 374. 375. 376. 377. 378. 379. 380. 381. 382. 383. 384. 385. 386. 387. 388. 389. 390. 391. 392. 393. 394. 395. 396. 397. 398. 399. 400. 401. 402. 403. 404. 405. 406. 407. 408. 409. 410. 411. 412. 413. 414. 415. 416. 417. 418. 419. 420. 421. 422. 423. 424. 425. 426. 427. 428. 429. 430. 431. 432. 433. 434. 435. 436. 437. 438. 439. 440. 441. 442. 443. 444. 445. 446. 447. 448. 449. 450. 451. 452. 453. 454. 455. 456. 457. 458. 459. 460. 461. 462. 463. 464. 465. 466. 467. 468. 469. 470. 471. 472. 473. 474. 475. 476. 477. 478. 479. 480. 481. 482. 483. 484. 485. 486. 487. 488. 489. 490. 491. 492. 493. 494. 495. 496. 497. 498. 499. 500. 501. 502. 503. 504. 505. 506. 507. 508. 509. 510. 511. 512. 513. 514. 515. 516. 517. 518. 519. 520. 521. 522. 523. 524. 525. 526. 527. 528. 529. 530. 531. 532. 533. 534. 535. 536. 537. 538. 539. 540. 541. 542. 543. 544. 545. 546. 547. 548. 549. 550. 551. 552. 553. 554. 555. 556. 557. 558. 559. 560. 561. 562. 563. 564. 565. 566. 567. 568. 569. 570. 571. 572. 573. 574. 575. 576. 577. 578. 579. 580. 581. 582. 583. 584. 585. 586. 587. 588. 589. 590. 591. 592. 593. 594. 595. 596. 597. 598. 599. 600. 601. 602. 603. 604. 605. 606. 607. 608. 609. 610. 611. 612. 613. 614. 615. 616. 617. 618. 619. 620. 621. 622. 623. 624. 625. 626. 627. 628. 629. 630. 631. 632. 633. 634. 635. 636. 637. 638. 639. 640. 641. 642. 643. 644. 645. 646. 647. 648. 649. 650. 651. 652. 653. 654. 655. 656. 657. 658. 659. 660. 661. 662. 663. 664. 665. 666. 667. 668. 669. 670. 671. 672. 673. 674. 675. 676. 677. 678. 679. 680. 681. 682. 683. 684. 685. 686. 687. 688. 689. 690. 691. 692. 693. 694. 695. 696. 697. 698. 699. 700. 701. 702. 703. 704. 705. 706. 707. 708. 709. 710. 711. 712. 713. 714. 715. 716. 717. 718. 719. 720. 721. 722. 723. 724. 725. 726. 727. 728. 729. 730. 731. 732. 733. 734. 735. 736. 737. 738. 739. 740. 741. 742. 743. 744. 745. 746. 747. 748. 749. 750. 751. 752. 753. 754. 755. 756. 757. 758. 759. 760. 761. 762. 763. 764. 765. 766. 767. 768. 769. 770. 771. 772. 773. 774. 775. 776. 777. 778. 779. 780. 781. 782. 783. 784. 785. 786. 787. 788. 789. 790. 791. 792. 793. 794. 795. 796. 797. 798. 799. 800. 801. 802. 803. 804. 805. 806. 807. 808. 809. 810. 811. 812. 813. 814. 815. 816. 817. 818. 819. 820. 821. 822. 823. 824. 825. 826. 827. 828. 829. 830. 831. 832. 833. 834. 835. 836. 837. 838. 839. 840. 84

10

1

1

22

• ٦٠

— 22 —

0

It will further be noted that the epidemic of Influenza in March, 1891, was not followed by an outbreak of Beri-Beri, or by any increase in the number of cases. This is of importance, as it is sometimes considered that Beri-Beri may exist in a latent form for prolonged periods, and appear only after some accident or disease which depresses the vital functions.

SECOND PERIOD, OLD GAOL AND NEW PUDOH GAOL, 1895 TO JULY 7TH, 1896.

This period is one of great importance, as the incidence of the disease amongst the prisoners confined in the two gaols varied greatly.

Most of the prisoners were transferred to the new gaol, and from May 23rd to the end of September there were no prisoners in the old gaol. During this period there was an epidemic at the new gaol of Cholera, with a mortality of over 50 per cent., and the first big epidemic of Beri-Beri commenced.

In September Beri-Beri cases were transferred to the hospital at the old gaol, and somewhat later healthy prisoners were also transferred to the old gaol.

Among the prisoners in the Pudoh Gaol, the general health from the first was unsatisfactory, and there was a good deal of Intestinal Disease as compared with the present amount. The hospital admissions for the more important Intestinal Diseases in 1895 are given in the subjoined table, and for comparison, 1902, a year in which the number of cases of Beri-Beri was the largest on record, is also given.

| 1895. | Pipe Water introduced from
Ampang Reservoir. | | | | | | | | | | | | Total. |
|---------------|---|------|------|------|------|-------|-------|------|-------|------|------|------|--------|
| | Jan. | Feb. | Mar. | Apr. | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | |
| Diarrhoea ... | 6 | 9 | 5 | 12 | 35 | 22 | 46 | 39 | 15 | 10 | 21 | 9 | 229 |
| Dysentery ... | 25 | 9 | 4 | 7 | 11 | 17 | 13 | 6 | 6 | 1 | 1 | 5 | 105 |
| Cholera ... | ... | ... | ... | ... | ... | ... | ... | 101 | ... | ... | ... | ... | 101 |
| 1902. | | | | | | | | | | | | | |
| Diarrhoea ... | 8 | 3 | 4 | 5 | 5 | 6 | 1 | 5 | 3 | 3 | 1 | 4 | 48 |
| Dysentery ... | 6 | 4 | 6 | 5 | 2 | 4 | 6 | 3 | 3 | 4 | 2 | 3 | 48 |

The water was supplied at first from surface wells, and, as in addition to the liability to faecal contamination, a large number of Chinese were buried in the vicinity, it was probably of inferior quality. Water from the Ampang Reservoir was substituted on August 21st, 1895, and the Cholera ceased immediately.

A total number of cases of Beri-Beri during this period was :—

| | Jan. | Feb. | Mar. | Apr. | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Total. |
|------|------|------|------|------|------|-------|-------|------|-------|------|------|------|--------|
| 1895 | ... | 1 | 1 | 2 | 3 | 7 | ... | 1 | 6 | 51 | 38 | 21 | 159 |
| 1896 | ... | 29 | 47 | 43 | 42 | 36 | 39 | 22 | ... | ... | ... | ... | 258 |

In point of time, the outbreak became important in September, immediately after the outbreak of Cholera and the change in the water supply. Before this time cases were in excess of the previous year. The six August cases had all commenced before the Cholera broke out, so that in the period before the Cholera there were 21 cases in eight months, as compared to three and two in the whole of the two previous years. It is not possible at this time to determine how many of these 21 cases were patients admitted with Beri-Beri, or infected previous to admission, but it can be ascertained in some, and of these one was admitted with the disease, four were admitted to hospital for Beri-Beri after less than one month's and two after less than two months'

incarceration. Three had the disease for six months or more, and four for three months or more before admission to hospital, so must have been admitted into gaol with the disease, or were relapsed cases.

In the remainder of the period the interval can be ascertained in the great majority of cases.

In prisoners sentenced for short periods, *i.e.*, six months and less, the period between conviction and admission to hospital for Beri-Beri, was under a week in 45, and under a month in 45 more, so that of the short-sentence prisoners, 90 had probably acquired the disease before admission to gaol, and 45 most certainly had done so out of the 141 of whom we have records.

The length of the period of residence in gaol amongst the short-sentence prisoners, before admission to hospital, is shown in the table :—

| | Under
1 week. | Under
1 month. | Under
2 months. | Under
3 months. | Over
3 months. |
|---------------------|------------------|-------------------|--------------------|--------------------|-------------------|
| 1895. | | | | | |
| January | 1 | ... | ... | ... | ... |
| February | ... | ... | ... | ... | ... |
| March | ... | ... | ... | ... | ... |
| April | ... | ... | ... | ... | ... |
| May | 1 | 1 | ... | 1 | ... |
| June | ... | ... | ... | ... | ... |
| July | ... | ... | ... | ... | ... |
| August | ... | 1 | 1 | ... | ... |
| September... .. | 2 | 6 | 15 | 4 | 3 |
| October | 2 | 3 | 4 | ... | 2 |
| November... .. | 5 | 5 | 1 | 1 | 1 |
| December | 4 | 5 | 1 | 1 | 2 |
| 1896. | | | | | |
| January | 12 | 2 | 4 | ... | ... |
| February | 2 | 3 | 1 | 2 | 1 |
| March | 3 | 5 | 1 | ... | 1 |
| April | 4 | 2 | ... | ... | 1 |
| May | 4 | 4 | ... | ... | 2 |
| June | 4 | 4 | 1 | ... | 1 |
| July | 1 | 4 | ... | ... | 1 |
| TOTAL | 45 | 45 | 29 | 9 | 15 |

Of the prisoners with longer sentences, *i.e.*, those convicted of more serious crimes or habitual criminals, during the same period, and in the first six months of their imprisonment, the period between conviction and admission to hospital for Beri-Beri was under one week in three, under one month in six, or nine in all, who had probably contracted the disease before admission. Three others developed Beri-Beri in less than two months, six in less than three months, and 32 in three months or more.

This epidemic was therefore associated both at the commencement and all through with an influx of large numbers of infected or diseased persons into the gaol, and in the early period there was comparatively little disease with certainty acquired in the gaol, though later, *i.e.*, after September, a large number of persons certainly acquired the disease in gaol, as they had been there over six months.

The cases of Beri-Beri admitted to hospital are divided into grades as follows :—those under six months including both short-sentence prisoners and those with longer sentences in the first six months of their imprisonment (*i.e.*, Lower Grade prisoners), Middle Grade who have served for more than six months, and Upper Grade who have passed through the Middle Grade and may have been years in prison. Relapses in gaol are considered separately.

| 1895. | Jan. | Feb. | Mar. | Apl. | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Total. |
|------------------|----------|----------|----------|----------|----------|------------|----------|----------|-----------|-----------|-----------|-----------|------------|
| S.S. & L.G. ... | 2 | 1 | ... | ... | 3 | ... | 3 | 3 | 42 | 20 | 14 | 20 | 108 |
| M.G. ... | ... | ... | ... | ... | ... | ... | ... | ... | 9 | 16 | 4 | 5 | 24 |
| U.G. | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | 1 |
| (?) ... | ... | ... | 2 | 3 | 4 | ... | ... | 3 | ... | ... | ... | ... | 12 |
| TOTAL ... | 2 | 1 | 2 | 3 | 7 | ... | 3 | 6 | 51 | 37 | 18 | 25 | 155 |

| 1896. | Jan. | Feb. | Mar. | Apl. | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Total. |
|------------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|------------|------------|------------|------------|------------|------------|
| S.S. & L.G. ... | 24 | 19 | 12 | 16 | 12 | 13 | 8 | ... | ... | ... | ... | ... | 104 |
| M.G. ... | 3 | 20 | 18 | 8 | 1 | 0 | 3 | ... | ... | ... | ... | ... | 53 |
| U.G. | ... | 2 | 6 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 9 |
| TOTAL ... | 27 | 41 | 36 | 25 | 13 | 13 | 11 | ... | ... | ... | ... | ... | 166 |

Old Gaol closed July 23rd, 1895.

All prisoners sent back to Pudoh.

In these returns I have excluded relapses in persons who had had an attack in gaol, as far as possible. This exclusion is necessary, as relapses may occur from trivial causes, but a large proportion are probably really, as regards causation, fresh attacks, and therefore, the occurrence of relapses cannot be ignored. The occurrence of relapses was as follows :—

| 1895. | Jan. | Feb. | Mar. | Apl. | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Total. |
|------------------|------------|------------|------------|------------|------------|------------|------------|------------|----------|----------|----------|----------|-----------|
| S.S., &c. ... | ... | ... | ... | ... | ... | ... | ... | ... | 5 | 1 | 1 | 4 | 11 |
| M.G. | ... | ... | ... | ... | ... | ... | ... | ... | 2 | 3 | 2 | ... | 7 |
| U.G. | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | 1 |
| TOTAL ... | ... | ... | ... | ... | ... | ... | ... | ... | 7 | 5 | 3 | 4 | 19 |

| 1896. | Jan. | Feb. | Mar. | Apl. | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Total. |
|------------------|----------|----------|----------|-----------|-----------|-----------|----------|------------|------------|------------|------------|------------|-----------|
| S.S., &c. ... | ... | 2 | 1 | 2 | 6 | 1 | 2 | ... | ... | ... | ... | ... | 14 |
| M.G. | 1 | 3 | 4 | 13 | 11 | 13 | 7 | ... | ... | ... | ... | ... | 52 |
| U.G. | ... | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | 2 |
| TOTAL ... | 1 | 5 | 5 | 15 | 18 | 15 | 9 | ... | ... | ... | ... | ... | 68 |

It is clear that there was in the early part of 1895 an importation of Beri-Beri into the gaol, and after July a great increase in the number of such cases.

During the whole course of the outbreak, a certain number of cases were from time to time admitted with the disease, or developed it so soon after their terms commenced, that they may be taken as being infected before admission, but as all the Middle Grade prisoners, 125, must have been more than six months in gaol and the 12 Upper Grade considerably longer, there can be no doubt that from September onwards the cause of the disease was in active operation in the gaol. At first, mainly Lower Grade prisoners were attacked, but later it was more prevalent amongst the Middle Grade.

Dr. McClosky, who was in charge of the gaol during the greatest part of the period, divided at the time the cases as follows :—

| | 1895. | 1896. | 1897. | 1898. |
|------------------------------|-------|-------|-------|-------|
| Contracted in gaol | 114 | 197 | (?) | 20 |
| Admitted with the disease... | 20 | 73 | (?) | 16 |
| Relapses | 7 | 213 | (?) | 20 |
| | <hr/> | <hr/> | <hr/> | <hr/> |
| | 141 | 483 | 97 | 56 |
| | <hr/> | <hr/> | <hr/> | <hr/> |

Probably Dr. McClosky's estimates are more correct, but as he included the length of time that the prisoners *stated* they had been sick before admission to hospital, I have preferred in my returns to give the interval between admission to gaol and hospital, both because many of the patients' statements cannot be trusted, and also so that the same principle of comparison can be applied to all the periods I am reviewing.

The actual differences in our figures are that for the period September to July. Dr. McClosky gives 69 as having contracted the disease outside, whilst according to the hospital returns 46 were admitted to hospital within a week and 49 more within a month of admission to gaol.

RELATION OF INCREASE IN BERI-BERI TO PROSPERITY AND CRIME.

It is of interest, therefore, seeing how closely connected admissions to prisons of persons in large numbers who have contracted the disease before admission is connected with this outbreak, to see what changes in either the laws or the administration of them, might have had any effect.

Interest naturally centres on the vagrants, as these form a large proportion of the short-sentence prisoners, and 45 out of the 46 cases which developed in less than two weeks were short-sentence prisoners.

Mr. Whitley, the Registrar of the Courts, has kindly supplied me with valuable information as to the changes which would affect the vagrant class that took place during this period.

He points out that there have been no substantial changes in the laws affecting vagrancy since 1893, when the Straits Ordinance of 1872 was adopted.

There have been important changes in the practice, dependent partly on the view taken by magistrates and partly on the energy and vigilance of the police.

In 1893, about 250 out of 2,825 persons brought before the Court were sent to gaol for vagrancy.

In 1894, between January 1st and October 22nd, only 12 were imprisoned for vagrancy, but between October 22nd and December 31st, 97 were so dealt with.

In 1895, between January 1st and April 30th, nine only were sent to the gaol as vagrants, in May there were 148, in June 21, and in July 81. In nearly all cases the sentence was one month, and between August 1st and December 31st 266 were sent to the gaol as vagrants, and in 1896, between January 1st to June 30th, 193 were so dealt with.

So that there was a great increase in the number of the convictions for vagrancy, commencing in May, 1895, and maintained all through 1895 and 1896, at more than twice the rate of the previous years.

Further, in 1893 and 1894, the sentences of the magistrates were usually small, 7 to 14 days, whilst in 1895 and 1896 the magistrates frequently inflicted

one month, two months', or four months' imprisonment, so that abundant time was allowed for the development of the disease and spread of infection.

There was a definite reason for the great increase in the convictions for vagrancy, as the staple industry of the country—tin-mining—was depressed, and many labourers were thrown out of employment in the mines, and, drifting to the towns, became vagrants, petty thieves, &c.

I am indebted to Mr. Lucas, Assistant Warden of Mines, for the information from which this statement and the subjoined tables are abstracted.

During 1893 and 1894, the price of tin varied considerably, but on the whole there was a downward tendency. The price fell more rapidly in 1895 and in 1896, and in consequence some mines were abandoned and no new ones were opened up. In the Report of the Mines Department for 1895, Selangor, it is stated that "No new mining centres were opened up. There seems to be a want of energy on the part of the Chinese to prospect for tin in the unopened country."

Price of Tin per pikul.

| | 1893. | | 1894. | | 1895. | | 1896. | |
|--------------|-------|------|-------|------|-------|------|-------|------|
| | Min. | Max. | Min. | Max. | Min. | Max. | Min. | Max. |
| January ... | 38½ | 39½ | 36½ | 37½ | 34½ | 36½ | 31½ | 32½ |
| February ... | 38 | 39½ | 36½ | 37½ | 35½ | 36 | 32 | 33½ |
| March ... | 39 | 39½ | 37½ | 39½ | 34½ | 35½ | 31½ | 32 |
| April ... | 38½ | 39½ | 37½ | 39½ | 34½ | 35½ | 31½ | 31½ |
| May ... | 36½ | 37½ | 39 | 40 | 34½ | 36½ | 31½ | 32½ |
| June ... | 36½ | 38 | 37½ | 39½ | 33½ | 34½ | 31½ | 32½ |
| July... | 36½ | 38 | 36½ | 37½ | 34½ | 36½ | 31½ | 32½ |
| August ... | 35½ | 38 | 25½ | 38 | 34½ | 35½ | 31½ | 33½ |
| September... | 36½ | 38 | 37½ | 39 | 34½ | 35½ | 32 | 32½ |
| October ... | 36½ | 38 | 36½ | 38½ | 34½ | 35½ | 31½ | 32½ |
| November... | 36½ | 37½ | 36½ | 37½ | 34½ | 35½ | 31½ | 32½ |
| December ... | 37½ | 38½ | 35½ | 37 | 33½ | 34½ | 31½ | 32 |
| AVERAGE ... | 37½ | 38½ | 35½ | 38½ | 34½ | 35½ | 31½ | 32½ |

As a proof of the exodus from mining districts, it may be stated that it was easy to obtain large numbers of Chinese Coolies for road work, &c., during the year.

Mr. Dykes, Senior Warden of Mines, gives me from his personal knowledge this further information, that "In the year 1893 the output of tin was 67,200 pikuls, valued at 23,000,000 dollars; in 1894 it was 79,200 pikuls, valued at nearly 29,000,000 dollars. These two years were said to be prosperous, and, as I can remember, Chinese drove about in their carriages and pairs, kept race-horses, and gave sumptuous banquets to Europeans.

"The year 1895 produced 82,100 pikuls, valued at 28,100,000 dollars, a decrease of 900,000 as compared with 1894. The year 1895 was a very bad one for miners, and I remember that there were many who failed to pay the wages due, and many who departed hurriedly to China on other business.

"The year 1896 saw the first decline in the output of tin since 1889. The output was 80,400 valued at 25,100,000, a decrease of 3,000,000 dollars. During 1895 and 1896 the Native States were suffering from severe depression."

In 1897, in a Report then written and published, Mr. Dykes further stated that "The year 1896 was a bad one for miners, owing to the low price of tin. The advancers failed to get back their money, and the Coolies were in many cases unable to obtain the full amount of money due to them. Those who had been miners forsook their vocations and were employed on road and railway construction." And again, in a Report Mr. Dykes was asked to write for the Tasmanian Government, he states that in 1896 "the price of food stuff was abnormally high, and this, coupled with the low price of tin, tempted many of those who had been miners to forsake their vocations and take to some other lucrative form of employment."

Mr. Dykes informs me that the first man to be turned out of a mine would be the sickly opium-smoking Coolie. The stronger ones could probably work harder and be able to earn their living.

The men who would be on the streets earning a precarious living by what they could pick up from someone else, would therefore be unhealthy, feeble, and decayed Coolies, who would stop at nothing to obtain opium to assuage their cravings.

1897 was also a bad year. The mining industry revived in 1898.

The question of vagrancy therefore became of greater importance, and it is to this depression in the value of tin that the influx of the poorer and worse fed and diseased classes into prison is to be attributed.

A Report of the Chief Police Officer, December, 1897, in this connection is very much to the point:—

"(1.) I do not anticipate any difficulty in catching vagrants. Their ultimate disposal is the point.

"(2.) In Perak the gaol authorities objected to them, and the State Surgeon got them transferred to hospital. Thence they absconded in a few hours, very often with a suit of hospital clothing.

"(3.) As fast as they are arrested they are back in the streets again, the result being a good deal of unproductive work for the Police and Courts Department.

"(4.) I am very sorry on behalf of this department that something cannot be done to keep these vagrants off the streets, the more especially as they are all petty thieves."

Mr. H. C. Ridges, Secretary for Chinese affairs, Selangor, supplied me with this and other references and information to the same effect as to the condition of the Chinese and vagrants in the years 1895—1897.

The reference to Perak is significant, as in Perak at the same time, *i.e.*, 1895 and 1896, there was practically no increase in Beri-Beri in the Taiping Gaol, though the depression must have been nearly as great in Perak as in Selangor.

The converse, however, occurs at Batu Gajah, a purely short-sentence prison. In that district the short-sentence prisoners, and particularly the vagrants, include the majority of the early cases of Beri-Beri, and as only the long-sentence prisoners are sent on to Taiping, a large proportion of the dangerous class, from the point of view of the introduction of Beri-Beri, are filtered off or excluded from the Taiping Gaol.

From Batu Gajah the number of transfers to Taiping Prison were in 1893, 1894, and 1895, respectively, only 83, 93, and 80, so that the mining vagrants from the Kinta district were effectually excluded.

CONDITION OF THE NEW (PUDOH) GAOL.

It must be remembered that during this period the works at the Pudoh Gaol were not complete. Much levelling, &c., of the ground was done during this period, and part of this ground was a Chinese graveyard. There is no evidence that many persons with Beri-Beri had been buried there, and certainly it was not used for burials of victims of any large epidemic of Beri-Beri. The site was not a sanitary one.

Further, the buildings were new, and were largely composed of cement, and therefore at first must be damp. The cells were badly ventilated, there were serious defects in the drainage, and a certain amount of leakage from the roof, and from pipes which convey the rain water from the roof, down the inside walls of the cells and under the lower tier of cells, occurred. These defects, subsequently remedied in part, may have contributed to the severity of the epidemic, and certainly would render the conditions unsuitable for persons suffering from the disease. As regards the dampness of the new building and disturbance of the ground as essential factors, it is well to note that the epidemic in 1902, though associated with a much lower death rate, affected a larger number of prisoners, though no disturbance of the ground took place, and the buildings were seven years old.

The series of observations tending to show that neither diet nor water is a factor in the causation of the disease was made by Travers and McClosky during this period. Naturally, the occurrence of an epidemic, first of Cholera and then of Beri-Beri, so soon after the new gaol was occupied, attracted much attention.

The disappearance of the Cholera, almost at once, after the substitution of water from the Ampang Reservoir, and the disuse of well water, occurred immediately before the outbreak of Beri-Beri became serious, and suggested the possibility that the town water was the cause of Beri-Beri.

EFFECT OF TRANSFER BACK TO THE OLD GAOL.

On September 12th, the prisoners with Beri-Beri were transferred back to the old gaol, and it was soon noticed that these prisoners recovered more rapidly than they had done in the new gaol, and that the death rate was much lower.

Dr. McClosky in his Annual Report for 1895, states that "at first the Beri-Beri patients were treated in the new gaol hospital with no beneficial results, and sooner or later died—rather sooner," and that the sick prisoners sent to the old hospital rapidly recovered.

Great care was taken and much attention was paid to the whole question of Beri-Beri, both by the State Surgeon, Dr. Travers, and Dr. McClosky, who daily visited the gaol. It was during this outbreak that Dr. McClosky noted that "one of the earliest, if not the very first symptom complained of, is a sense of oppression and fulness in the epigastrium after even the smallest quantity of food."

Partly in order to remove as many as possible of the prisoners from the infected area, over 100 healthy prisoners free from Beri-Beri, were transferred

on October 25th, to the old gaol, and no cases of Beri-Beri occurred amongst the prisoners so transferred during the whole period up to July 9th, 1896, when those still undergoing their sentences were sent back to the old gaol.

The number was not, however, maintained, as in 1896 the numbers at the old gaol for the first half-year only averaged 109, including Beri-Beri patients and convalescents sent from the new gaol.

The prisoners in the old gaol consisted of three parties :—

(1.) Prisoners who had had Beri-Beri in the new gaol, and were transferred whilst ill to the old gaol hospital. These varied in 1896 from a monthly average of 16 to 31½.

(2.) A party working inside the gaol at rotan work, and composed of convalescents from the old gaol hospital, and others transferred whilst healthy from the new gaol. These would be long-sentence prisoners. Average monthly numbers of those working inside the gaol varied from 29 to 54½, but the number who were not Beri-Beri convalescents cannot be ascertained.

(3.) Short-sentence prisoners engaged in extra-mural work. The number varied from 37 to 54.

The average for the first six months of 1896 was, in hospital, 20 ; engaged in intra-mural work, 43 ; extra-mural work, 46.

No cases of Beri-Beri occurred amongst the third party, and only relapses amongst the two other parties.

As controls, only the third party and the unknown number of the second party, who were free from Beri-Beri when transferred to the old gaol, are to be considered. These prisoners were all first admitted to Pudooh Gaol, and then, after a variable period, transferred to the old gaol. Before the transfer they were again medically examined, and none were transferred who showed any signs or symptoms of Beri-Beri.

The short-sentence prisoners, according to Mr. Kydd, then the Gaoler, probably included none sentenced to less than three months.

As a test of "ground," "earth," or "place" infection as opposed to personal infection, the "control" in the experiment was not a sound one, as the selection of prisoners and exclusion of those with Beri-Beri from the parties sent to work at the old gaol makes a real difference.

The comparison of the high incidence in the Pudooh Gaol with the immunity of the prisoners in the old gaol is not invalidated as a test of the effects of water or food by this selection of persons, and exclusion of persons suffering from Beri-Beri in the early stages.

An observation of interest was made in connection with the water supply. On April 1st, the water from the Ampang Reservoir to the new gaol was diverted to the old gaol, and at the new gaol the use of the well water was resumed.

This change did not result in any diminution in the amount of Beri-Beri in the new gaol, nor did any cases originate in the old gaol.

On July 1st, the Ampang Reservoir water was again used in the new gaol.

Other Institutions, using the same rice, are considered by Dr. Travers as further controls, and to a great extent they were so, but each must be considered separately, as the conditions were not uniform, and in these, the

Decrepit Vagrants Ward and the Leper Asylum differed to an important extent from those in either of the gaols.

The actual diet in the Vagrant Ward and in the Leper Asylum was rather less liberal than that in the gaol, but as little or no work was done, relatively, was probably at least equal. In the gaol, prisoners are not allowed to change their rations or to differently apportion them. No such provisions occur at the other Institutions. Both the vagrants and lepers may supplement their diets by presents, begging, sale of products of their work or other property as they are able to do so. More frequently, than purchasing food, they procure tobacco or opium.

These differences, in my opinion, reduce the value as "controls" of the inmates of these Institutions.

Further, the strict confinement, rigid discipline, and constant monotonous work of the gaol differ in moral and mental effect from the surroundings in the other Institutions.

It cannot be urged, assuming the disease to be caused by a toxine or poison contained in the rice, either that it would necessarily effect a larger proportion of weakly and debilitated persons, than of robust and active persons, or otherwise. We know, as a matter of fact, that robust active persons do suffer severely from Beri-Beri. It can, however, be considered that as "controls," this difference in general health of the two classes introduces another factor. It is also to be remembered that the Lepers and incurables have, on the whole, been long exposed to the worst conditions of life, and that a large proportion must have previously been exposed to such conditions as are likely to produce Beri-Beri, so that they represent possibly an immune residuum, and therefore their value as "controls" is further reduced. As "controls," therefore, only a part of those working at the old gaol can be considered as satisfactory.

The prisoners belonged to various grades, and were employed on industrial work, with the exception of the parties employed extra-murally.

The work did differ in the two gaols, and was lighter in the old gaol. No importance can, in my opinion, be attached to the differences in the class of work, and for the purpose of comparing the effect of diet in the production of Beri-Beri, the "controls" were a sound test, though, if the relative numbers at different periods of service in the two institutions had been kept, it would have been more satisfactory, as both Middle and Upper Grade prisoners, as well as Lower Grade and short-sentenced prisoners were employed in both.

As bearing on the theories of food infection, such slight differences as were present in the conditions were not of vital importance. Though inferior as "controls" to the inmates of the old gaol, the immunity of the inmates of the Leper Asylum and Incurable Wards is further evidence to the point, as showing that rice is not the essential agent in the production of Beri-Beri. It must be remembered that, exclusive of relapses, the majority of cases in the new gaol were amongst short-sentence prisoners.

No cases at all originated in the gaol, though the food supplied was the same in the two institutions, and for a period of four months was all from the same kitchen.

THIRD PERIOD—PUDOH GAOL, 1896-1905.

During the third period, various observations have been made and published by Travers, Hamilton Wright and Durham. The food has been varied from time to time, and certain changes made in cooking and serving the food. Rice, "Rangoon," has been supplied all the time by the same contractor. This rice is also supplied to the inmates of the Leper Asylum, of the Tai Wah or destitute ward, both Institutions free from Beri-Beri, and to the District Hospital, where cases of Beri-Beri originate. Throughout the whole period cases of Beri-Beri occurred with certain intervals, and at times assumed epidemic proportions. The absence of Beri-Beri from other Institutions supplied with the same rice is of value, but as the condition of the patients in those Institutions differs, they are not, as controls, conclusive.

Table I. gives the number of admissions and deaths during the whole period up to July, 1905. These figures include relapses :—

| | 1896. | | 1897. | | 1898. | | 1899. | | 1900. | | 1901. | | 1902. | | 1903. | | 1904. | | 1905. | |
|-------------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|-------------|---------|
| | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. | Admissions. | Deaths. |
| January ... | ... | ... | 31 | 8 | 4 | ... | 22 | 2 | 12 | ... | 20 | ... | 68 | 4 | 22 | 1 | ... | ... | 8 | 1 |
| February | ... | ... | 15 | 5 | 3 | ... | 10 | 1 | 15 | ... | 9 | ... | 42 | 7 | ... | ... | 2 | ... | 5 | ... |
| March | ... | ... | 24 | 3 | 2 | ... | 9 | ... | 15 | 1 | 49 | 1 | 62 | 3 | ... | ... | 5 | ... | 6 | ... |
| April | ... | ... | 3 | 1 | 17 | ... | 10 | ... | 7 | 1 | 21 | 2 | 80 | 3 | ... | ... | 1 | ... | 6 | ... |
| May | ... | ... | 10 | 3 | 1 | ... | 1 | ... | 5 | ... | 11 | ... | 93 | 6 | ... | ... | ... | ... | 1 | ... |
| June | ... | ... | 17 | 1 | 1 | ... | ... | ... | 3 | ... | 15 | 1 | 83 | 5 | ... | ... | 3 | ... | 1 | ... |
| July | ... | ... | 12 | 1 | 5 | ... | ... | ... | 11 | ... | 5 | ... | 67 | 7 | ... | ... | 6 | ... | ... | ... |
| August | 15 | 1 | 35 | 2 | 6 | 1 | 1 | ... | 16 | ... | 18 | ... | 76 | 2 | ... | ... | 3 | ... | ... | ... |
| September | 50 | 8 | 35 | 6 | 1 | ... | 2 | ... | 20 | 2 | 12 | ... | 131 | 7 | ... | ... | 2 | ... | ... | ... |
| October | 45 | 2 | 35 | 8 | 1 | ... | 11 | 4 | 17 | ... | 11 | 1 | 99 | 5 | ... | ... | 3 | ... | ... | ... |
| November | 65 | 3 | 51 | 16 | 10 | ... | 3 | ... | 21 | 1 | 16 | ... | 35 | 2 | ... | ... | 1 | ... | ... | ... |
| December | 45 | 5 | 7 | ... | 22 | 1 | 4 | ... | 38 | 2 | 18 | ... | 55 | 3 | ... | ... | 6 | ... | ... | ... |
| TOTAL | 220 | 19 | 275 | 54 | 73 | 2 | 73 | 7 | 180 | 7 | 205 | 5 | 891 | 54 | 27 | 2 | 32 | ... | 27 | 1 |

TABLE I.—Admissions and Deaths from Beri-Beri in Pudooh Gaol, from August, 1896, onwards.

TABLE II.

1st attacks in Gaol. Relapses or attacks in Persons with a history of Beri-Beri outside the Gaol, are not excluded.

| | 1896. | 1897. | 1898. | 1899. | 1900. | 1901. | 1902. | 1903. | 1904. | 1905. |
|---------------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|
| January ... | ... | 17 | 3 | 15 | 11 | 13 | 51 | 16 | ... | 5 |
| February ... | ... | 8 | 2 | 6 | 14 | 7 | 38 | ... | ... | 2 |
| March ... | ... | 12 | ... | 7 | 11 | 15 | 48 | ... | 3 | 1 |
| April ... | ... | 4 | ... | 6 | 6 | 17 | 66 | ... | ... | 3 |
| May ... | ... | 4 | 1 | ... | 7 | 8 | 73 | ... | ... | ... |
| June ... | ... | 4 | 1 | ... | 2 | 8 | 61 | ... | 2 | ... |
| July ... | ... | 4 | 4 | ... | 11 | 2 | 53 | ... | 3 | ... |
| August ... | 6 | 17 | 5 | 1 | 15 | 11 | 59 | ... | 2 | ... |
| September ... | 24 | 13 | ... | 1 | 18 | 9 | 83 | 1 | 2 | ... |
| October ... | 32 | 20 | 2 | 8 | 18 | 8 | 80 | 1 | 3 | ... |
| November ... | 35 | 30 | 8 | 3 | 20 | 8 | 23 | 3 | 1 | ... |
| December ... | 22 | 7 | 12 | 3 | 30 | 13 | 45 | ... | 6 | ... |
| TOTAL ... | 119 | 140 | 38 | 50 | 163 | 119 | 680 | 21 | 22 | 11 |

TABLE III.

| | Awaiting Trial and Simple Imprisonment. | Under 6 months S.S. & L.G. | Over 6 months M.C. & U.G. |
|-----------|---|----------------------------|---------------------------|
| 1896 ... | 1 | 61 | 41 |
| 1897 ... | 4 | 81 | 54 |
| 1898 ... | ... | 25 | 13 |
| 1899 ... | ... | 22 | 26 |
| 1900 ... | ... | 111 | 49 |
| 1901 ... | 1 | 85 | 29 |
| 1902 ... | 27 | 496 | 157 |
| 1903 ... | 3 | 20 | ... |
| 1904 ... | 2 | 11 | 15 |
| 1905 ... | ... | 2 | 9 |
| TOTAL ... | 38 | 914 | 393 |

It will be seen from these tables that the outbreak continued throughout the greater part of 1897, that it was still in excess of the rate in the old gaol in 1898 and 1899, and in the early part of 1900, even allowing for the increase in the number of prisoners.

It became more serious in the later months of 1900, and continued at a high rate all through 1901, and in 1902 there was a great increase, followed early in 1903 by a rapid cessation of the outbreak. Since then, the rate has been almost as low, in proportion to the number of prisoners, as in the years before 1895.

Table III. shows the occurrence of Beri-Beri in those under and over six months' residence in the prison, and also the number of cases amongst those awaiting trial, and undergoing simple imprisonment, who are in common

association wards. It will be seen that a considerable number of the first attacks in gaol occurred amongst prisoners who had been over six months incarcerated, and these had certainly acquired the disease in gaol, though some of them, no doubt, had had previous attacks outside.

Some of the other cases, those under six months, had certainly acquired the disease before admission, as they were sent to hospital in a week or less, and others, those sent to hospital in less than a month, in all probability also brought the disease with them.

TABLE IV.

| | Short-sentence Prisoners. | | | | | Long-sentence Prisoners = Lower Grade. | | | | |
|-------------|---------------------------|----------|-----------|-----------|----------------|--|----------|-----------|-----------|----------------|
| | 1 Week. | 1 Month. | 2 Months. | 3 Months. | Over 3 Months. | 1 Week. | 1 Month. | 2 Months. | 3 Months. | Over 3 Months. |
| 1895 | 15 | 21 | 22 | 6 | 8 | 1 | 3 | 1 | 3 | 13 |
| 1896† | 30 | 24 | 7 | 2 | 7 | 3 | 7 | 6 | 6 | 32 |
| TOTAL ... | 45 | 45 | 29 | 8 | 15 | 4 | 10 | 7 | 9 | 45 |
| 1896† | 9 | 11 | 6 | 1 | 10 | ... | ... | ... | ... | ... |
| 1897 | 7 | 8 | 11 | 6 | 20 | ... | 4 | 1 | 4 | 17 |
| 1898 | 5 | 9 | 2 | ... | 2 | ... | 1 | 1 | 1 | 4 |
| 1899 | 3 | 2 | 1 | 1 | 6 | 1 | 2 | ... | ... | 5 |
| 1900 | 6 | 12 | 16 | 19 | 31 | ... | 2 | 1 | 3 | 16 |
| 1901 | 2 | 9 | 13 | 12 | 18 | ... | 3 | 4 | 5 | 12 |
| 1902 | 3 | 24 | 44 | 68 | 121 | 5 | 14 | 28 | 31 | 102 |
| 1903 | 3 | ... | 2 | ... | 8 | ... | ... | ... | 1 | 7 |
| 1904 | 5 | 2 | 1 | ... | 1 | ... | 1 | 1 | 1 | 1 |
| 1905 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| TOTAL ... | 43 | 77 | 96 | 107 | 217 | 6 | 27 | 36 | 46 | 165 |
| GRAND TOTAL | 88 | 122 | 125 | 115 | 232 | 10 | 37 | 43 | 55 | 210 |

* The Epidemics were associated with an increase in the number of those admitted into gaol in the early stage of Beri-Beri, i.e., those admitted to hospital with definite Beri-Beri a few days after conviction, and of those who developed Beri-Beri within a month of admission. This is significant. The men are either in the incubation stage, in the prodromal stage, or infected immediately after admission, i.e., whilst in the association ward, where they are confined whilst awaiting trial, for the first night after sentence, or when undergoing simple imprisonment. It is to be noted that this increase occurs both in the early part and all through a prolonged epidemic.

The facts certainly point to an introduction of the disease from outside, and to infection of prisoners subsequently, directly or indirectly, from these admissions, and to maintenance of the epidemic by the fresh introduction of the disease.

It will be observed that there is an enormous variation from time to time in such admissions; and a comparison of the two classes of prisoners, i.e., those with short sentences, convicted of vagrancy, petty theft, and other crimes, and those with longer sentences convicted of more serious crimes, is made in Table IV.

This table shows the total incidence of the disease during the first months of imprisonment in short-sentence prisoners, vagrants and the like, and in long-sentence prisoners, during the last 10 years.

It will be seen, that amongst the short-sentence prisoners, no less than 210 developed the disease so soon that they probably had acquired the disease before admission, and possibly some of the 125 who were admitted to hospital between one and two months after admission to gaol, had also acquired the disease before admission to gaol.

Amongst the long-sentence prisoners in the same period, only 47 belong to the first class, with a possibility that some of the 43 who developed the disease in two months, were also infected before admission.

After the experience of Batu Gajah (short-sentence prisoners), where, out of 65 prisoners who developed Beri-Beri in less than one week, from 1901 to 1904, 44 had been imprisoned for vagrancy, suspicion naturally falls on the vagrants; and the association of the outbreak at Kwala Lumpur in 1895, with an increase in the number of convictions and more severe sentences for vagrancy, supports this view.

It must be admitted, however, that there are certain points in connection with vagrants that point to only certain classes of them being the real source of danger.

- (1.) In the decrepit ward at the Kwala Lumpur Hospital, Beri-Beri rarely occurs, nor does it in the Leper Asylum.
- (2.) During one of the periodic raids on vagrants in the present year, 161 were sent to gaol between May 1 and July 31st, one of these only showed signs of Beri-Beri, or developed it within one month; 29 gave a previous history of that disease, or 18 per cent. Of 373 other prisoners, admitted in the same period, only 34 had a previous history, or 9 per cent.

As has been shown elsewhere, comparatively few cases of Beri-Beri occur amongst the residents in Kwala Lumpur. Most of the cases come from the surrounding mining districts, and in 1895 the fall in price of tin caused the abandonment of many workings and an increase in vagrancy, and an influx of the vagrants from the mines into the town, where, either by begging, by petty theft, or otherwise, they were able to obtain a meagre existence.

In Batu Gajah, the mass of vagrants are from the surrounding mines.

It is therefore to the external circumstances, which affect the occasional, rather than the habitual criminal, that the variations in the amount of Beri-Beri introduced into the prison are to be attributed.

As far as the occurrence of Beri-Beri after 1896 is concerned, a consideration of these tables shows that the variations were such that the period can be conveniently subdivided into seven unequal periods or terms, and that these are the same whether the total number of cases or the number exclusive of relapses is considered.

These periods or terms are arbitrary divisions, made solely on the basis of the varying number of cases which occurred in the gaol, in order to see what relation existed between occurrence of a large or small number of cases and other factors.

A close relationship is to be observed between the number of cases which occurred in the gaol, and the number of persons admitted in the early stages of the disease, whether these were short-sentence prisoners, as in 1895 and 1896, or persons awaiting trial, as in 1902.

The first period is that from August, 1896, to November, 1897. This period is a continuation of the outbreak of 1895, though the number of cases was smaller.

In addition to any sources of the disease in the gaol, cases were introduced, as in the previous period, but not in such great numbers.

The length of residence, in these different terms, before the admission to hospital of S.S. prisoners for Beri-Beri, is shown in Table A.

TABLE A.—Period No. 3. August, 1896-1905.

| | Under
1 week. | Under
1 month. | Under
2 months. | Under
3 months. | Three
months
and over. |
|-----------------------------------|------------------|-------------------|--------------------|--------------------|------------------------------|
| 1. Aug., 1896, to Nov., 1897 ... | 16 | 19 | 16 | 7 | 28 |
| 2. Dec., 1897, to Nov., 1898 ... | 4 | 8 | 2 | ... | ... |
| 3. Dec., 1898, to April, 1899 ... | 4 | 3 | 1 | 1 | 5 |
| 4. May, 1899, to Dec., 1899 ... | ... | ... | ... | ... | 2 |
| 5. { 1900 | 4 | 18 | 17 | 15 | 29 |
| 1901 | 2 | 9 | 13 | 12 | 18 |
| 1902 | 3 | 24 | 44 | 68 | 121 |
| Jan., 1903 | ... | ... | 2 | 1 | 2 |
| 6. Feb. to Aug., 1903 | ... | ... | ... | ... | ... |
| 7. Sept., 1903, to June, 1905 ... | 8 | 2 | 3 | ... | 2 |
| TOTAL | 41 | 83 | 98 | 104 | 207 |

FOR COMPARISON, PERIOD No. 2—1895 TO JULY, 1896.

| | Under
1 Week. | Under
1 Month. | Under
2 Months. | Under
3 Months. | Three
Months
and over. |
|------------------------------|------------------|-------------------|--------------------|--------------------|------------------------------|
| January, 1895, to July, 1896 | 45 | 45 | 29 | 9 | 15 |

As these periods are unequal in length, for purposes of comparison, the monthly average for each period is appended in Table B, together with the average rate per month of admissions to the hospital for Beri-Beri, and of the occurrence of the disease amongst prisoners awaiting trial.

TABLE B.

| | Average
Number
of Cases
Monthly. | Awaiting
Trial or
Simple
Imprison-
ment. | Average Number of Prisoners developing
Beri-Beri in less than | | | |
|-------------------------------|---|--|--|----------|-----------|-------------------|
| | | | 1 Week. | 1 Month. | 2 Months. | Over
2 Months. |
| 1. Aug., 1896, to Nov., 1897 | 30.5 | .33 | 1.06 | 1.7 | 1.3 | 4.4 |
| 2. Dec., 1897, to Nov., 1898 | 4.8 | ... | .33 | .75 | .25 | .65 |
| 3. Dec., 1898, to April, 1899 | 14.6 | ... | 1 | 1 | .4 | 2.2 |
| 4. May to Dec., 1899 | 3.6 | ... | ... | ... | ... | .5 |
| 5. { 1900 | 15 | ... | .33 | 1.66 | 1.48 | 5.2 |
| 1901 | 17 | .08 | .17 | 1 | 1.41 | 3.9 |
| 1902 | 74.25 | 2.25 | .66 | 3.16 | 6.1 | 18.5 |
| 1903 | 22 | 1 | ... | ... | 3 | 8 |
| 6. Feb. to Aug., 1903 | ... | ... | ... | ... | ... | ... |
| 7. Sept., 1903, to June, 1905 | 2.7 | .13 | .36 | .14 | .18 | .29 |

In the last four columns, long-sentence prisoners who developed the disease in the periods stated are included, as well as the short-sentence prisoners.

It must be remembered that prisoners, either awaiting trial or undergoing simple imprisonment, are kept in the admission (association) ward.

Assuming that all prisoners awaiting trial, and all who were admitted to hospital in less than one month, had contracted the disease outside, it follows therefore, from the above table, that these may be grouped together.

TABLE C.

| | | | | Average admitted
per month
already infected. | Average cases
per month
developing in gaol. |
|--------|-----|-----|-----|--|---|
| Term 1 | ... | ... | ... | 3.09 | 30.5 |
| " 2 | ... | ... | ... | 1.38 | 4.8 |
| " 3 | ... | ... | ... | 2 | 14.6 |
| " 4 | ... | ... | ... | ... | 3.6 |
| " 5 | ... | ... | ... | 3.1 | 35.4 |
| " 6 | ... | ... | ... | ... | ... |
| " 7 | ... | ... | ... | .63 | 2.7 |

The conclusion based on these coincidences is confirmed by comparison with other prisons.

It may be urged that, so far from showing the disease is brought from outside and spreads amongst the prisoners, the gaol epidemics are only part of a general epidemic, affecting equally those in the gaol, new arrivals, and the general public.

A comparison of the gaol figures with the returns of admission to hospitals for Beri-Beri, and of deaths attributed to this disease, shows that this is not the case.

It will be seen that, in all but the 4th and 7th terms (both short ones, seven and eight months respectively), cases that had previously contracted the disease were admitted into the gaol. That the larger the number of such admissions, the larger the number of monthly admissions to hospital for Beri-Beri amongst the other inmates. The occurrence, though only at a low rate, of cases in the gaol during the term No. 4, shows that, when introduced, the infection may, under gaol conditions of cleanliness, still continue to spread to a slight extent, but some of these at least, might have been relapses in persons who had had Beri-Beri before.

The last term appears to be very similar to the normal condition of affairs at Taiping, a few admissions in the early stage of the disease, a few cases originating in the gaol, and a few relapses. The ventilation of the cells is now good. Extra-mural work has been discontinued. The work for short-sentence prisoners, and during the first year for long-sentence prisoners, is now mainly stone-breaking, but for a few months from March 9th, 1904, they were engaged in extensive excavation and levelling, inside the prison walls. There has been no change in the diet except for the period May to December, 1901, during Dr. Hamilton Wright's experiment.

The majority of cases of Beri-Beri that have originated in the gaol of late have been of an unusually mild type.

These tables show that, as in 1895—1896, there is a close relation between the number of admissions with the disease and the number of cases originating in the gaol.

During the second and fourth terms the rate was singularly low, and, associated with the rise in those admitted with the disease, there was a corresponding increase in the number of cases.

In the fifth period, there was a steady rise in the number of cases, though in neither 1900 or 1901 were the numbers much more than half that in 1896 and 1897.

The number of admissions of persons who had probably contracted the disease outside was also considerably less than in those years. It was during this period that Dr. Wright's observations were made.

In 1902 there was the worst outbreak known. The average number of cases is more than twice that of any previous year. The number of prisoners who were admitted who had probably contracted the disease outside, was 6·07 per month ; more than twice as many as in any previous period.

This increase is not as in previous outbreaks due mainly to vagrants, but was composed of 27 prisoners awaiting trial, 19 long-sentence prisoners, and 27 short-sentence prisoners. The main excess above the average therefore was in the long-sentence prisoners, and persons of the same class awaiting trial, whilst there was only a slight increase in those convicted of vagrancy and petty crime.

Of the prisoners awaiting trial, not one was from Kwala Lumpur itself ; they were all from purely mining districts ; and of the long-sentence prisoners only two were from Kwala Lumpur, the others were from mines, mainly those from near Rawang and K. Kubu.

In 1900—1904 the conditions were very different to those in the epidemic of 1895. The price of tin was high, though fluctuating.

| | | |
|-------------------------------|---------|------------|
| In 1900 the average price was | \$74 | per pikul. |
| In 1902 it was | \$80·6 | " |
| In 1903 it was | \$83·65 | " |

The cost of extraction had increased so much that the mine owners, early in 1902, attempted to enforce a seven-hour working day. This resulted in riots and strikes amongst the miners, and much general lawlessness amongst these classes, including gang robberies.

It was from the inhabitants of these mining districts that the majority of persons, who developed Beri-Beri whilst awaiting trial or shortly after conviction, came.

Early in 1903 a similar attempt to enforce longer hours was made, and resisted peaceably by the miners. The tribute system of payment, as a compromise, has been adopted in most of the larger mines, and works, if not economically, at any rate without friction.

It was on observations made at this gaol during 1900, 1901 and 1902, that the results and conclusions arrived at by Dr. Hamilton Wright and Dr. H. E. Durham were founded.

Of these experiments, one only dealt to any great extent with the diets. For a period of eight months, May to December, 1901, all the prisoners received 6 ozs. meat, and no fish was given.

The average rate, 13·2 per mensem, was less than in 1900, 15 per mensem, and much less than in the first four months of the same year 27·25, and far less in the following twelve months, 74·3.

If relapsed cases are excluded, the rate for these eight months was 7·3 against 13·6, and 13 for 1900 and the first four months of 1901, and 57·7 of 1902. The reduction in the amount of Beri-Beri during this period may be due to the improved diet, but as so many still occurred, fish as *the* essential cause or absence of sufficient animal food as *an* essential cause, can be definitely excluded.

Dr. Wright's results are published in full in "Studies from the Institute of Medical Research," Vol. II., Nos. 1 and 2.

The points he desired to determine were :—

- (1.) Whether the disease was contracted intra-murally or extra-murally by prisoners engaged on work beyond the gaol walls.
- (2.) The value of certain diets as etiological factors.
- (3.) Whether or not a racial immunity existed.

To determine the first point, Dr. Wright divided the prisoners into four parties : one 101 strong, had all had Beri-Beri either in or out of the hospital within the previous five years. They occupied "D" hall and worked rotan in Shed "A."

The second gang, 113 strong, had no signs of Beri-Beri, and gave no history of any previous attack. They occupied halls "C" and "B" and worked in sheds "B" and "C."

A third gang, of 33, who had not had Beri-Beri, occupied the administration block, and their work was entirely extra-mural. Any vacancies in this gang were filled from party No. 2.

The fourth gang, of 38, were employed as cooks, tailors, &c. They had had Beri-Beri in the gaol, and had recovered completely.

All prisoners admitted were, if they had had Beri-Beri, sent to party No. 1, others to No. 2, or sometimes No. 3.

Of party No. 1 (old cases of Beri-Beri), daily average 128·8, 184 had relapses or fresh attacks in the eleven months.

Of party No. 2 (no previous history), daily average 221·64, 90 cases occurred.

Of party No. 3 (no previous history), daily average 34, no cases of Beri-Beri occurred.

Of party No. 4 (old gaol cases), daily average 41, nothing more than slight recurrences.

Parties Nos. 1 and 2 were constantly recruited from outside.

Party No. 3 was usually recruited from No. 2.

Party No. 4 was recruited only from prisoners who had been in gaol for a prolonged period.

The case incidence of Beri-Beri in the parties was, therefore—No. 1, 144 per cent., but this includes relapses, &c., as well as fresh attacks.

No. 2, 40·7 per cent., first attacks only, as any person who contracted Beri-Beri was transferred to No. 1.

No. 3, Nil.

No. 4, slight recurrences only.

Owing to the frequency with which, after prolonged intervals relapses occur, it was impossible to determine how many of the cases in party No. 1 really originated in the prison, though probably the majority did so.

Party No. 2 included by accident, in some instances, persons who had had Beri-Beri, and developed a recurrence. In such cases the patients were transferred, on recovery, to party No. 1, and are not included in the returns for party No. 2.

TABLE D.—Table of Occurrence of Cases.

| | 1901. | | | | | | | | 1902. | | |
|-----------------|-------|-------|-------|------|-------|------|------|------|-------|------|------|
| | May. | June. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Jan. | Feb. | Mar. |
| Party No. 1 ... | 18 | 4 | 5 | 9 | 15 | 22 | 33 | 35 | 24 | 10 | 9 |
| " " 2 ... | 1 | 1 | 2 | 3 | 8 | 8 | 11 | 18 | 25 | 11 | 2 |

Dr. Hamilton Wright considers that his observations afford proof :—

(1.) That Beri-Beri is independent of diet considered as diet, since the diet was as physiologically correct from May to December, 1901, as that in the Japanese Navy after 1884; moreover, whilst taking this diet, 49 cases originated, and 125 re-contracted the disease or relapsed, whilst after December, when the regular and lower scale of diet was reverted to, there was no increase, but rather a decline in the number of cases of Beri-Beri.

There was, however, an enormous increase after this period, according to medical records.

(2.) That the gaol itself is a focus in which the virus of Beri-Beri is generated. He considers that the freedom of party No. 3 proves that it was not due to volatile poison or suspended micro-organisms wafted in the air. He proved that the rice was sterile when delivered to the prisoners. Fish was excluded from the dietary, and the preparation of the vegetables was such as to destroy all micro-organisms and known toxins. The freedom of party 3, who were on the same diet, again excludes diet qualitatively as a factor in the production of Beri-Beri.

The only external factor that was not eliminated was the introduction of the disease in the clothes or systems of prisoners during the eleven months that the experiment lasted.

Even in party No. 2 several cases of Beri-Beri, relapses in old Beri-Berics, were accidentally admitted. Further, in his analysis of the length of incarceration of the 90 cases originating in gaol amongst this party, he shows that Beri-Beri developed as follows :—

| No. of }
Cases } | 1-7
days. | 7-14
days. | 14-21
days. | 21-30
days. | 30-45
days. | 45 dys. to
2 mths. | 2-3
mths. | 3-4
mths. |
|---------------------|--------------|---------------|----------------|----------------|----------------|-----------------------|----------------|--------------|
| | 3 | 10 | 6 | 6 | 9 | 10 | 11 | 12 |
| No. of }
Cases } | 4-5
mths. | 5-6
mths. | 6-7
mths. | 7-8
mths. | 8-9
mths. | 9-12
mths. | 12-18
mths. | |
| | 8 | 6 | 2 | 1 | 1 | 2 | 3 | |

Dr. Hamilton Wright appears to assume that the period of incubation is a short one of ten days or so. Even if this is occasionally the case, it, in my

opinion, is so rarely among newly imported coolies, as at Sungei Lembing, under a month, and the onset in some of the cases is so gradual that I consider it more probable nearly all the cases originating in the first month, and some of those in the second month, possibly, had contracted the disease before admission, or on the first night of admission, in the association ward, where they are kept one night before the medical examination.

According to this view some 25 out of the 90 introduced the disease in their own persons, into party No. 2. The freedom of party No. 3, a small party of 34, is probably to be explained by the rarity of admissions direct from outside.

It is clear, however introduced, that cases did subsequently originate in the gaol itself in persons who had been too long in confinement to have possibly been infected before admission.

Among other points of interest, Dr. Hamilton Wright shows that cases originated in all parts of the gaol occupied by party No. 2. The incidence in the different floors was :—

| | C. Hall. | B. Hall. | Right Centre. | Total. |
|-----------------------|----------|----------|---------------|--------|
| 1st Tier | 11 | 11 | ... | 22 |
| 2nd and 3rd Tiers ... | 31 | 18 | 19 | 68 |
| TOTAL ... | 42 | 29 | 19 | 90 |

The C. Hall was that most constantly occupied, which, he considers, probably accounts for the difference. As 22 cases in these two halls developed on the first floor, and 49 in the two upper tiers together, an average of 24.5 in each tier, the increased elevation and fresher air in the upper tiers made no appreciable difference.

As regards relation of immunity to general health, 83 out of the 90 who developed Beri-Beri were classed as "robust."

Dr. Wright further shows that of the 90 who developed Beri-Beri, 77 were Chinese, 8 were Malays, and 2 were Indians.

As, of the prisoners who formed the original party No. 2, or passed through it, 1902 were Chinese, 94 were Malays, and 220 were Indians, the figures show that there was no racial immunity, though they may indicate a diminished susceptibility in the case of the Indians.

For full details of these experiments, the reader is referred to Study, Vol. II., No. 1, from which the above is in the main abstracted.

After the cessation of Wright's experiment, the division into gangs was no longer maintained, and there was a great increase in the amount of Beri-Beri. All grades were affected, and the total for the year exceeded that in any previous year.

No change was made in the diets, in the work of the prisoners, or in the routine from that in the last month of Dr. Wright's experiment till September.

The outbreak subsided suddenly in January, 1903. If first attacks only are considered and relapses excluded, the subsidence is seen to be more gradual, as the first attacks, August to October, averaged 28.7 per month,

and November to January (4, 11, 6), only 7 per month. The changes in the period were, that from September 23rd, the prisoners were employed in extra-mural work, and took their food, except in the evening, outside the cells. From October 20th, the Ranji (morning rice) was cooked at 3.30 a.m., and served hot instead of being cooked on the preceding evening.

Between January and May, 1903, the ventilation of the cells was greatly improved.

Some of these measures would diminish the liability to infection, particularly to infection of food by means of dust, &c., in the cells.

Hamilton Wright claims that a disinfection of the cells with Formalin spray in February is to be regarded as one of the important causes of the diminution. The proportion of the gaol disinfected is uncertain. Dr. Hamilton Wright is of opinion that only a small portion was not so treated, about $\frac{1}{4}$ th. The statements of the gaolers are that $\frac{1}{4}$ th or less was so treated. No records were kept in the gaol on this point, and therefore the evidence in both cases is based on memory. In any case, this disinfection was followed by an increase in Beri-Beri for over six months. The other measures for disinfection of cells—the use about twice a year of perchloride of mercury, was continued, but had been practised previously. He is in error in considering that any change affecting the majority of prisoners in the disposal of the dejecta took place. The prisoners all passed their motions in their cells, if so inclined, between 4.30 p.m. and 5.30 a.m., the only change was that prisoners in solitary confinement were allowed to pass their evacuations *during the day* outside instead of, as previously, inside the cells.

How far all or any of the changes were responsible for the diminution in the number of fresh infections is doubtful, but it is clear that the spread of the disease amongst the prisoners in the gaol diminished towards the end of 1902, and it is probable that, to some extent at least, the extra-mural work in the open sheds was an important factor. Another factor, however, was that there was a marked diminution in the number of persons admitted with the disease or in the period of incubation, as show in Table E.

TABLE E.—Kwala Lumpur Gaol.

| | 1902. | | | | | | | | | | | | 1903. |
|--|-------|------|------|------|------|------|-------|------|-------|------|------|------|-------|
| | Jan. | Feb. | Mar. | Apl. | May. | Jun. | July. | Aug. | Sept. | Oct. | Nov. | Dec. | Jan. |
| Awaiting Trial ... | 2 | 2 | 7 | 4 | 5 | 2 | 1 | ... | ... | 1 | ... | ... | 1 |
| Simple Imprisonment | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| Long-Sentence Prisoners under 1 month | ... | ... | 2 | 4 | 4 | 4 | 1 | 2 | ... | 1 | ... | ... | ... |
| Short-Sentence Prisoners under 1 month | 4 | 5 | 2 | 2 | 3 | 1 | 1 | 1 | 2 | 1 | 2 | 3 | ... |
| TOTAL ... | 7 | 7 | 11 | 10 | 12 | 7 | 3 | 3 | 2 | 3 | 2 | 3 | 1 |

The diminution was mainly in the prisoners awaiting trial and in long-sentence prisoners.

In the following seven months no such cases were admitted, and no cases developed in the gaol.

CONCLUSIONS.

The various changes in Kwala Lumpur Gaol have proved conclusively :—

- (1.) That Beri-Beri is independent of the water supply.
 - (2.) That fish, fresh or salted, is not responsible for the disease.
 - (3.) That changes in diet are not associated with any decided effect, and that, at the most, a slightly more liberal diet only reduces the rate.
 - (4.) That thorough cooking of the food has no effect in the prevention of Beri-Beri.
 - (5.) That amongst different gangs of prisoners kept distinct, as in Traver's experiment in 1895-96, at the old and new prisons, and in Wright's in 1902, between parties Nos. 2 and 3, two bodies of men may be partaking of the same mass of rice, cooked and distributed in the same way, and yet no Beri-Beri may occur in the one party, and a great deal in the other. The number of inmates of the old gaol, controls, in Traver's experiment, over 45, and the number forming party No. 3. controls, in Wright's experiment, average 34, are not quite large enough to serve as absolutely conclusive controls, as in some, the most favourable exposure to the necessary conditions required to produce Beri-Beri, three months, was not attained.
 - (6.) That the one constant factor in prison Beri-Beri is the admission of cases in the early stages of Beri-Beri to the gaol. That such admissions in numbers are usually followed by an outbreak of Beri-Beri ; but that the epidemic tends to die out unless there is a continued introduction of fresh cases.
- Conversely, large outbreaks of Beri-Beri in the prisons are invariably preceded and accompanied by the admission of cases of Beri-Beri, in the early stage, from outside.
- (7.) The disease once introduced, may spread widely, and is little affected by external circumstances, but appears to be slightly affected, both as regards frequency and severity, by changes in work, in diet, and possibly in ventilation, and improvements in other hygienic conditions.

On considering carefully the figures in these prisons, it is difficult, either when each institution is taken by itself, or when they are all considered together, to avoid the conclusion that the disease is introduced by persons in the early or incubation stage of the disease, and that from such persons the disease spreads, directly or indirectly, to others of the prisoners. That such spread of the disease is limited, and under the ordinary gaol conditions of cleanliness and discipline, the outbreak only becomes serious when a constant stream of imported cases is taking place.

The different incidence in the gaols or elsewhere, is shown in the table following :—

| | Singapore Prison. | Lunatic Asylum, Singapore. | Penang Gaol. | Kwala Lumpur Gaol. | Taiping Gaol. | Batu Gajah Gaol. | Kwala Lipis Gaol. | Pekan Gaol. | Sungei Lembing Mines. | State Hospitals, Selangor (2). | |
|-------|-------------------|----------------------------|--------------|--------------------|---------------|------------------|-------------------|-------------|-----------------------|--------------------------------|---------|
| | | | | | | | | | | Cases. | Deaths. |
| 1893 | ... | ... | ... | 2 | ... | ... | ... | ... | ... | 3829 | 894 |
| 1894 | ... | 58 | ... | 3 | ... | ... | ... | ... | ... | 2817 | 683 |
| 1895 | ... | 128 | 3 | 159 | ... | ... | ... | ... | ... | 3781 | 711 |
| 1896 | 3 | 194 | 9 | 478 | 16 | ... | ... | ... | ... | 5518 | 990 |
| 1897 | 3 | 123 | 3 | 275 | ... | ... | 13 | 8 | ... | 4884 | 1066 |
| 1898 | 124 | 155 | 3 | 73 | ... | 51 | 10 | ... | ... | 1870 | 244 |
| 1899 | 165 | 121 | 1 | 73 | 11 | 89 | 4 | 2 | 132 | 1837 | 333 |
| 1900 | 224 | 153 | 3 | 180 | 23 | 107 | 8 | 5 | 230 | 2428 | 390 |
| 1901 | 219 | 102 | 1 | 205 | 16 | 149 | 2 | 3 | 203 | 2065 | 311 |
| 1902 | 415 | 99 | 2 | 891 | 42 | 73 | 1 | 4 | 37 | 2673 | 325 |
| 1903 | 169 | 21 | ... | 26 | 13 | 23 | 5 | ... | 151 | 1912 | 351 |
| 1904 | 266 | 154 | 4 | 32 | 33 | 91 | 2 | ... | 222 | 2083 | 326 |
| TOTAL | 1588 | 1308 | 29 | 2397 | 154 | 583 | 45 | 22 | 975 | 35697 | 6624 |

At Christmas Island, in 1902, there were 693 cases, with 89 deaths ; in 1903, 591 cases—60 deaths ; in 1904, 973 cases—91 deaths ; and in 1905 (up to September), 392 cases—6 deaths.

In Kwala Lumpur and Taiping the average strength has been steadily rising, but was rarely over 500 till the last few years. Batu Gajah is peculiar as a purely short-sentence prison. Average strength is under 300.

Penang is also a short-sentence prison, but the prisoners with sentences up to one year are left there. Average strength over 300. The two other prisons are smaller : under 100, and about 25 respectively. In both these, prisoners are to some extent engaged in extra-mural work.

The main comparison therefore is between Kwala Lumpur and Taiping. Both are mainly supplied from mining districts, and the incidence of the disease, prior to 1895 and subsequent to 1902, was much the same in both.

The big epidemic in Kwala Lumpur of 1895 and 1896, was only represented at Taiping by some 16 cases. As this was a time of severe depression in the mines, owing to the fall in the price of tin, the influx of cases into Kwala Lumpur is easily accounted for. The steps taken by the State Surgeon, Perak, as recorded by the Chief Police Officer, probably prevented an influx and epidemic in the Taiping Gaol, similar to that which took place in Kwala Lumpur.

Since 1898 the Batu Gajah Gaol has received the prisoners from the largest mining district, and has, therefore, absorbed the more important mining vagrant class. Even of the long-sentence prisoners, who are transferred to Taiping, they are not always transferred at once, and so those in the incipient stages develop the disease at Batu Gajah, and not at Taiping.

This short-sentence prison acts as a receiving house, and filters off from the Taiping Gaol the majority of the mass of prisoners who, in Kwala Lumpur, have infected the gaol.

The discipline, work and diet vary slightly in the different prisons. At Penang, Kwala Lumpur, Taiping, and Kwala Lipis, Rangoon rice has been constantly used. It is supplied to each place by different contractors. At Batu Gajah, "Siamese" rice prepared in Penang, and grown mainly in Province Wellesley, is used.

There are no facts to show that there is any earth or place infection, unless it be admitted that, by such infection, we mean that for a short period after the occupation of a place by diseased persons enough of the *materies morbi* remains to infect other persons. The facts are more in favour of indirect or intermediate conveyance of the disease from one person to another, than of any direct contagion.

One of the best instances of so-called house infection I have seen is that recorded by Dr. Fletcher, amongst the people who have lived at No. 10, Pudoh Road, during 1904.

The first case occurred early in March, in a man who had been living at the house since January 15th, *i.e.*, about six weeks.

He, his wife and family, all left the house about the middle of April.

The second case was a man who occupied the house in May. Beri-Beri appeared in June, and he then left for Malacca. The third case was a man living with No. 2, and he developed Beri-Beri about a month later.

The fourth patient had been living in the house since June 26th, and the first symptoms of Beri-Beri were noticed on July 20th, after less than one month's residence.

These four people were all Malacca Portuguese, and they are the only known cases of Beri-Beri that have developed in the last two years amongst the 150 to 200 of this race resident in the town. They used "Siam" rice No. 2, and it was bought at various Chinese shops.

It will be noted that the first case developed after about six weeks' residence, the second and third after one and two months' residence respectively, and the fourth after less than one month.

Subsequent occupants have not acquired the disease. The house is one of a row of houses mainly occupied by Government and other clerks, Tamils or Eurasians. The houses are separate, and have separate kitchens and latrines. At the back of the row, between the house and kitchen, is a badly graded drain, common to the whole row, but as there were no other cases of Beri-Beri in the row during the period, the source of infection must have been in the house.

It is not, in my opinion, necessary to assume place or house infection, as of the four cases three were living together in the house, and they developed the disease successively, after intervals which are quite compatible with infection from person to person. The period between the first case leaving the house and the second entering it seems to have been only two or three weeks; so that this case might have easily derived the infection from the previous occupant, or from articles he left behind him.

The series supports the view that the disease is communicated indirectly from man to man, rather than that there is a place or house or earth infection.

As regards the means of infection, it appears that continued introduction from without, of fresh cases, is necessary for the continuation of an outbreak in a gaol, but not as will be shown in a mine. Failing such introductions into a gaol, the outbreak soon diminishes and dies out.

This suggests that the ordinary routine cleanliness and hygienic surroundings of the inmates of the gaol are not favourable for the prolonged spread of the disease. The conditions induced by the fresh arrivals are more so.

The system of keeping the prisoners for one or two nights in a common cell may bear an important part in the spread of the disease. This cell at Kwala Lumpur had a wooden floor. It is brushed out daily, and the floor is washed once a week. Care has to be exercised and little water used, as there are rooms underneath, and if much water be used these rooms are flooded.

The prisoners have a bath on admission to the gaol ; those awaiting trial wear their own clothes, the others are provided with a prison uniform.

Each prisoner makes use of a blanket and mat placed on the floor. These blankets and mats are exposed to the sun, but not washed, and as they may be used by six or seven different prisoners each week, the opportunities for the spread of any contagious disease or of any disease spread by vermin are such that even a slightly contagious disease might readily assume epidemic proportions.

The various lock-ups in which prisoners may be detained for a night or two also fall under suspicion.

Although, as I think, the figures quoted show that much of the disease originates in prisoners during the early months of their imprisonment, and that in this way the disease is introduced, other cases originate so long after admission that it is certainly spread in the gaol itself as well.

There are two ways, apart from continuity of buildings, in which it spreads from one group to another : first, by the transfer of prisoners from one grade to another ; secondly, by relapsed cases.

I am inclined to think that the first, *i.e.* the transfer of prisoners in the incubation stage of the disease from one grade to another, is the most important. Prisoners are not medically examined on being transferred from one grade to another. The number of relapses varies so much with the number of fresh cases, that many of them may be the result of reinfection. All, however, are not. In the Taiping Gaol in 1898, the only man affected had three relapses, at a time when no other cases occurred, and when no cases were admitted with the disease. The experience in the same gaol of the period October, 1899, to April, 1900, when all the fresh cases were in prisoners who had been over a year in the prison, and when no prisoners were admitted with the disease, was preceded and associated with a number of relapses in other prisoners, who had been over a year in the gaol and were of the same grades as the prisoners attacked for the first time.

This points to the relapsed cases as a source of infection.

It has been urged that the prison outbreaks of Beri-Beri are only part of a general epidemic. Little support is given to this view by a com-

parison of the incidence, in different Institutions, with the numbers admitted to the State Hospitals.

As regards Selangor, there is a certain correspondence between the admissions for Beri-Beri to the State Hospitals and the incidence of Beri-Beri in the gaol, and there is no doubt that the prosperity or otherwise of the country has an influence on the amount of disease and the class of crime. 1895, 1896 and 1897 were the years in which there were the largest number of admissions and deaths in hospital from Beri-Beri. These were the years in which there was much distress amongst the miners, from the depression in the value of tin.

A larger proportion would no doubt seek hospital relief than in more prosperous times, but it can, I think, be admitted that there was a real increase in the amount of Beri-Beri. These three years were also the years of the first epidemic in the Kwalar Lumpur Gaol.

In 1898 when the tin industry revived there was a great diminution in the amount of Beri-Beri in the gaol, and also in the admissions to State Hospitals for Beri-Beri.

Since then the fluctuations in the amount of Beri-Beri in the gaol have been large, and of admissions to hospital have been small.

A general prevalence of Beri-Beri outside, no doubt leads to a larger number of the admissions to gaol of persons with Beri-Beri, and so the other inmates of the gaol in turn suffer. The conditions of prison life, other than this increased liability to infection by new arrivals, are not affected by poverty outside the prison.

In the consideration of the effect of variations in the staple industry of the country—tin-mining—on the incidence of a disease, the effects will differ in the admissions to hospital or to prison in different ways, though indirectly, poverty will increase both.

The question is complicated, as in Malaya not only has the main product—tin—varied greatly in value, but the currency—silver—has also varied in value.

For the labouring classes, the silver value affects the prosperity more than the gold value.

The employers of labour are more affected by the gold value.

The output of tin, approximately, is a measure of the number of men employed in the mines.

The economic history, as regards these factors, is graphically represented in the chart abstracted from the Annual Report of the Resident General, Sir William Taylor, K.C.M.G., for 1904. [*See Charts 1 & 2.*]

It will be seen that there was a steady rapid increase in the output of tin to 1894, and a slower increase in 1895, indicating an influx of labour to the mines.

The gold value was stationary till 1893, when there was a rapid decline, but the silver value remained stationary till 1895, when it commenced to fall, and did not rise above the average till 1898.

These three years showed an increase in the admissions to the hospitals for Beri-Beri, and include the period of the first big epidemic in the Kwala Lumpur Gaol.

From 1898 onwards, there was a rapid increase in the silver value of the exported tin, and up to 1900 a corresponding increase in the gold value, associated with an increased output, but not up to the mark of 1895.

Since 1900, with fluctuations, there has been a fall in the gold value, although the output of tin has increased, and therefore the expenditure on labour. The increase in the number of labourers has been greater, nearly twice as great as the increase in the output, as mines are worked to a greater depth than when tin was less valuable.

Such a state of affairs has necessarily pressed heavily on the employers, to whom the gold value is of most importance, but has not so much affected the labourers, as the silver value has increased *pro rata* with the output.

During the period, the admissions to hospital for Beri-Beri have shown little variation, but the number of cases of Beri-Beri in prison has shown an enormous increase, culminating in 1902 in the largest outbreak known, and of cases introduced into the gaol from the mining districts, partly as a result of illegal resistance (criminal) on the part of the miners, to an attempt on the part of the employers to obtain longer hours of work from their labourers.

In 1903 there was an increase in the gold as well as in the silver value of tin; and of even more importance, after a drawn battle unattended with violence between the employers and labourers, an amicable working arrangement was arrived at.

RECOMMENDATIONS.

That, as Beri-Beri is introduced into prisons mainly by the short-sentence prisoners, vagrants and the like, in the cases where an enlargement of a prison is necessary, short-sentence prisoners be confined in a separate block or institution.

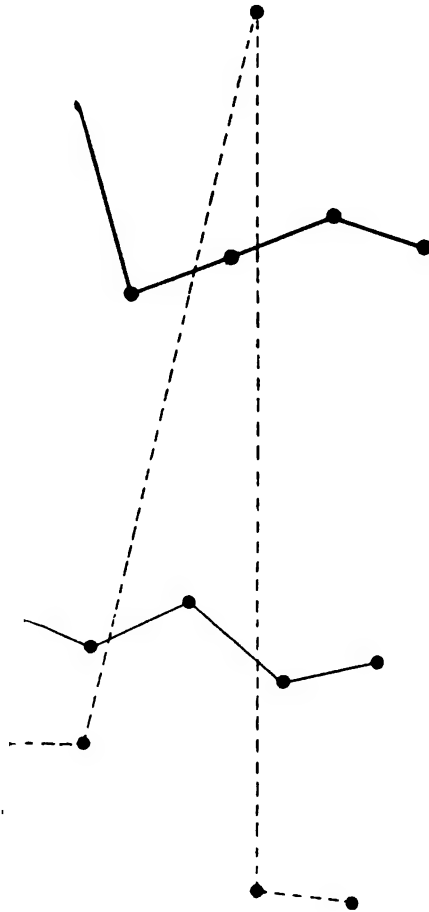
(2.) That the confinement in an association ward for the first night of all prisoners should be discontinued. That prisoners awaiting trial, those undergoing simple imprisonment, and convicted prisoners awaiting medical examination should be kept absolutely separate.

(3.) That a mat and blanket should be served out to each prisoner on admission to the gaol at the same time as his clothes, and that this mat and blanket should be taken with the prisoner on any change of cells.

(4.) That the cells, whether association or otherwise, in which prisoners are confined on admission to prison, should be such as to enable thorough daily cleansing, and such disinfectants as may be considered suitable after occupation.

(5) That every effort should be made to destroy vermin on the prisoners or in their cells. That bed-boards should be as free from cracks as possible, and that they should be locked down so as to be capable of removal, thorough washing, and exposure to verminicides.

1901. 1902. 1903. 1904.



in the gold value of Tin (1894-1898) was associated
 mission rate for Beri-Beri, in both the Hospitals
 when the silver value remained high and the gold
 there was an increase in the prison rate only

(6.) That no cell in which a case of Beri-Beri has developed should be again occupied till it has been thoroughly disinfected, and the mat and blanket used should be destroyed.

(7.) That all blankets should be disinfected after use, before being returned to the store, and that those in use should as a routine be sterilized or washed at regular intervals.

In the Kwala Lumpur, Taiping, Batu Gajah and Penang Prisons, the outbreaks of disease were associated in most cases with the introduction from outside of cases of the disease in the early stage.

In all, it may be said that no outbreaks occurred in periods when no such introduction took place, with the exception of Taiping Gaol, from October, 1899 to April, 1900 ; in that case it was associated with a large number of relapses.

Further, though it is not contended that the disease once thus introduced does not continue to spread, yet, that this spread is not indefinite, and that without the continued introduction of fresh cases from outside, an outbreak declines and dies out, as that in Kwala Lumpur in 1899 and 1903, that in Penang in 1905, in Taiping, May, 1897 to June, 1899, broken only by three relapses in one prisoner, &c., as can be seen from the various tables.

Instances are apparent in which the introduction of cases has not been followed by any outbreak, and it is fully admitted, that under ordinary conditions, the disease does not necessarily spread from one person to another.

I have emphasized the point that the danger appears to be from fresh cases admitted to a prison and developing the definite secondary symptoms—the nervous symptoms—shortly after admission. Relapses appear to have been responsible for the small outbreak in Taiping in 1899. This is certainly exceptional. Relapses undoubtedly do occur after considerable intervals, without any apparent possibility of mediate or intermediate infection ; but in the great majority of prisons, relapses have occurred at a time when fresh cases are also occurring, and have often ceased during periods when no cases are admitted with the disease, or originated in the gaol. The frequent periods when there have been no cases in Taiping, and the rarer ones in the other prisons, are instances of this.

To recapitulate. The prison evidence tends to show that outbreaks of Beri-Beri in these institutes are started by antecedent cases of the disease, usually in recently admitted prisoners, rarely by relapses in prisoners who have been long in gaol.

Further, that the introduction of persons with the disease, or in the earlier stages of the disease, is a concomitant of the continuance of such outbreaks.

As regards other factors, particularly food, relation of outbreak to season or weather, and the mode of infection if the disease be infectious, these matters will be considered subsequently.

OCCURRENCE IN OTHER CLASSES.

Outbreaks in mines, towns, and other communities, are more difficult to investigate, as the conditions are variable and the movements of people are not under much control. Conditions of food, work, exposure, and association

are largely dependent on individual caprice in most of the mining communities, where the disease is or has been prevalent.

As a general rule, in the Federated Malay States the disease does not occur in native villages where the people are living under their ancestral conditions, growing their own food, and having little or no contact with the Chinese. It does occur amongst Malays engaged as police, watchmen, or boatmen, who mix more with other races, and buy their own food usually imported from Chinese shops.

It does not occur, except in rare instances, amongst Tamils, whether of the better class who are employed as clerks, &c., or amongst the coolie class, whether working in mines or on plantations.

In the Federated Malay States, it is mainly a disease of the Chinese, and affects the coolie class, whether employed in the mines or in agriculture, more than the well-to-do Chinaman.

The inhabitants of the towns suffer less than those of the country, though to this rule Singapore is an exception.

The new arrivals suffer most severely, and the Straits-born Chinaman very little.

In prison epidemics there is comparatively little difference in the incidence of the disease, when the relative numbers are considered, between the individuals of different races, if only the attacks certainly acquired in gaol are considered.

Europeans and Eurasians, even in prison, are rarely attacked.

There is hardly a place or district where Chinese are found in which Beri-Beri does not occur. In the mining districts, the history is fairly uniform; and a district may be opened for some time and no Beri-Beri may occur, but when it does it rapidly becomes prevalent, and is attended with a high mortality; but after a varying period, though cases still originate in the district in greater numbers than in the towns, they are far less numerous and less severe than during the earlier outbreaks. In certain mines the disease in a severe and epidemic form, though varying in prevalence from time to time, is, for a prolonged period, the constant cause of most of the mortality and sickness.

Of such communities, the mining districts on the east coast of the Malay Peninsula may be taken as examples. These bodies of men are practically isolated from those on the west coast, but communicate to a considerable extent with Singapore. The variations in the season are, as will be seen from the rainfall, greater than in Kwala Lumpur, and are greater also than in Singapore, Penang, or any places on the West Coast of the Peninsula.

MINES.

The two main mining districts on the east coast are Sungei Lembing and Sungei Blat. On the former, the number of coolies employed has not varied very greatly, and may be taken as about 1,200, and of these some 40 per cent. are Sinkeh, either employed by the Company, which imports them, directly, or in numbers up to nearly two-thirds sublet to contractors.

In the other district, Sungei Blat, the numbers employed, nearly all free men, have been rapidly increasing. They were estimated as about 500 in 1899 and 1900, 800 in 1901, 1,000 in 1902, 2,000 in 1903, and now are nearly 3,000.

In the Sungei Lembing District, in the last six years, 790 deaths from Beri-Beri are reported to have occurred, which gives an average of 132, or 110 per 1,000, from this cause alone. The incidence was heavy in 1903 and 1904, as seen from the table, but even in the lowest year recorded (1902), amounted to 37, or, for that year, over 40 per 1,000.

In Blat, cases have occurred in each year, but not only is the total number, 209, much smaller, and smaller proportionately, but in no year has the death rate been so high. In 1903, the total deaths from Beri-Beri were 59, say 30 per 1,000, as against 144, or 120 per 1,000 in Sungei Lembing. The year 1903 was one of the worst in the records of both districts.

The variation in the seasons is much more marked on the East Coast than on the West. Not only is the rainfall much greater in the winter months, as shown in the table, but the temperature is lower, and there are strong cold winds. In fact, there is a marked contrast between one monsoon and the other, instead of the small difference which exists in Selangor.

RAINFALL.

| KUANTAN. | | | KWALA LUMPUR. | | |
|------------------|-------|-------|------------------|-------|-------|
| | 1902. | 1903. | | 1902. | 1903. |
| January | 16.22 | 11.46 | January | 3.77 | 4.41 |
| February | 20.21 | 10.82 | February | 10.24 | 3.06 |
| March | 4.25 | .35 | March | 9.65 | 11.73 |
| April | 14.36 | 5.57 | April | 11.36 | 9.22 |
| May | 7.04 | 3.19 | May | 11.28 | 10.82 |
| June | 2.20 | 6.01 | June | 4.55 | 6.03 |
| July | 2.59 | 4.46 | July | 2.98 | 0.20 |
| August | 4.65 | 5.57 | August | 4.91 | 9.58 |
| September | 9.76 | 5.69 | September | 8.60 | 5.54 |
| October | 6.59 | 14.95 | October | 20.89 | 9.79 |
| November | 7.49 | 13.88 | November | 18.07 | 5.91 |
| December | 9.68 | 10.35 | December | 10.10 | 13.60 |

It will be noted in the returns for Beri-Beri, both of admissions to Kuantan Hospital, and of deaths from Beri-Beri in the district, that in December, January, February and March, the number of cases is much higher than in the dry months.

**TABLE A —Cases of Berl-Berl, Sungel Lembing Hospital,
Sinkhs only, exclusive of relapses.**

| | 1898. | 1899. | 1900. | 1901. | 1902. | 1903. | 1904. | Total. |
|------------------|-----------|------------|------------|------------|-----------|------------|------------|------------|
| January ... | ... | 17 | 19 | 39 | 5 | 8 | 31 | 119 |
| February ... | ... | 26 | 58 | 31 | 4 | 12 | 35 | 166 |
| March ... | ... | 11 | 16 | 31 | 4 | 8 | 20 | 90 |
| April ... | ... | 12 | 6 | 21 | 2 | 4 | 9 | 54 |
| May ... | ... | 20 | 20 | 16 | 1 | 9 | 7 | 73 |
| June ... | ... | 9 | 12 | 23 | 2 | 7 | 8 | 61 |
| July ... | ... | 4 | 7 | 10 | 1 | 10 | 1 | 33 |
| August ... | 2 | 8 | 10 | 1 | ... | 4 | 5 | 28 |
| September ... | 3 | 5 | 9 | 5 | 3 | 11 | 10 | 43 |
| October ... | 3 | 3 | 9 | 1 | ... | 10 | 19 | 42 |
| November ... | 8 | 9 | 17 | 1 | 1 | 8 | 16 | 52 |
| December ... | 13 | 8 | 25 | 2 | 7 | 38 | 22 | 102 |
| TOTAL ... | 29 | 132 | 208 | 181 | 30 | 129 | 183 | 863 |

Coolies only.

| | 1900. | 1901. | 1902. | 1903. | 1904. | Total. |
|------------------|-----------|-----------|----------|-----------|-----------|------------|
| January ... | ... | 5 | 3 | 3 | 4 | 15 |
| February ... | ... | 6 | 1 | 1 | 2 | 10 |
| March ... | ... | 3 | ... | 2 | 9 | 14 |
| April ... | ... | 1 | 1 | 1 | 2 | 5 |
| May ... | ... | ... | 1 | 1 | 1 | 3 |
| June ... | ... | 1 | ... | ... | 1 | 2 |
| July ... | 2 | 2 | ... | 3 | 1 | 8 |
| August ... | 2 | 2 | ... | 1 | 1 | 6 |
| September ... | 6 | ... | ... | 1 | 5 | 12 |
| October ... | 4 | ... | ... | 2 | 4 | 10 |
| November ... | 6 | 1 | ... | 1 | 2 | 10 |
| December ... | 2 | 1 | 1 | 6 | 7 | 17 |
| TOTAL ... | 22 | 22 | 7 | 22 | 39 | 112 |

TABLE B.—Deaths from Beri-Beri, Kwala Kuantan and Sungei Lembing.

| | 1899. | 1900. | 1901. | 1902. | 1903. | 1904. | Total. |
|------------------|-----------|------------|-----------------|-----------|------------|-----------------|---------|
| January ... | 8 | 12 | 22 | 18 | 9 | 45 | 114 |
| February ... | 15 | 14 | 27 | 6 | 8 | 33 | 103 |
| March ... | 13 | 15 | 14 | 3 | 14 | 27 | 86 |
| April ... | 9 | 11 | 26 | 2 | 12 | 28 | 88 |
| May ... | 12 | 18 | 27 | 4 | 12 | 10 | 88 |
| June ... | 5 | 9 | 26 | 3 | 12 | 10 | 65 |
| July ... | 7 | 11 | 14 | 5 | 7 | 7 | 51 |
| August ... | 7 | 14 | 6 | 2 | 11 | 4 | 44 |
| September ... | 4 | 10 | (?) | 2 | 12 | .. | over 28 |
| October ... | 6 | 10 | (?) | 1 | 15 | 6 | " 38 |
| November ... | 3 | 8 | (?) | 3 | 11 | over 16 | " 41 |
| December ... | 4 | 6 | (?) | 3 | 21 | (?) | " 34 |
| TOTAL ... | 93 | 138 | over 162 | 52 | 144 | over 186 | |

Blat Cases (all sent to Kwala Kuantan Hospital).

| | 1899. | 1900. | 1901. | 1902. | 1903. | 1904. | Total. |
|-----------------------|----------------|----------------|----------------|-----------------|-----------------|-----------------|--------|
| January ... | ... | ... | ... | 1 | 2 | 24 | 27 |
| February ... | 1 | ... | ... | ... | 1 | 21 | 23 |
| March ... | ... | ... | ... | ... | 3 | 11 | 14 |
| April ... | ... | 1 | ... | ... | 6 | 7 | 14 |
| May ... | ... | 2 | (?) | 1 | 7 | 1 | 11 |
| June ... | 1 | 6 | (?) | 1 | 3 | 6 | 17 |
| July ... | 2 | 10 | (?) | ... | 5 | 7 | 24 |
| August ... | ... | 17 | (?) | ... | 3 | 7 | 27 |
| September ... | ... | 2 | (?) | ... | 3 | 9 | 14 |
| October ... | 2 | 1 | (?) | 2 | 7 | ... | 12 |
| November ... | 1 | 7 | (?) | 2 | 10 | ... | 18 |
| December ... | 3 | 6 | (?) | 3 | 9 | ... | 21 |
| TOTAL ... | 9 | 52 | ... | 10 | 59 | 93 | |
| POPULATION ... | (?) 500 | (?) 500 | (?) 800 | (?) 1000 | (?) 2000 | (?) 2800 | |

A reference to the admissions at Sungei Lembing Hospital shows also the same tendency to an increase in the number of cases during the wet season, and so also do the small numbers in the Pekan Gaol. There is a decided difference in the incidence of the disease in these places, corresponding to the changes of the season, instead of a tendency to an increase in the amount which is the most that is indicated in the returns from the West Coast.

BERI-BERI AT SUNGEI LEMBING.

Much of the work on Beri-Beri has been in connection with the origin and progress of an outbreak amongst susceptible people, when the disease is introduced amongst them. Such work is well exemplified by the histories of the outbreaks of Beri-Beri in the Kwala Lumpur Goal and elsewhere.

A gaol is peculiarly suited for such enquiries, as the conditions of life are uniform and under control. During my stay in the country, there has been nothing that could be called an epidemic in the gaol at Kwala Lumpur, as for seven months there were no cases, and subsequently, though cases have occurred, they have, exclusive of recurrences and persons admitted with or developing the disease in a few days, been only 22, an average of one a month, which could, with any degree of probability, be attributed to intramural conditions at Kwala Lumpur.

Some information can be obtained from enquiries into the course of an outbreak amongst gangs of susceptible persons introduced into an area where the disease is present. In the stages of an epidemic in a gaol, where cases are numerous, each new admission is usually a susceptible person introduced into such an area; but more information can be obtained from a consideration of the fate of gangs introduced as labour into a district where the disease is endemic, although the conditions of life vary more, and are more complicated than in gaol.

The mines and works of the Pahang Corporation, Limited, at Sungei Lembing were taken as a good sample of the fate of such gangs. This place was selected in accordance with advice given me by Dr. Lucy, State Surgeon, Pahang.

The advantages of this district were—(1) That a large number of cases of severe Beri-Beri occurred; (2) That a large number of Sinkehs were annually imported; and (3) That, owing to the inaccessibility of the place, complications from contact with other gangs of labour were few, and that the foodstuffs for all the gangs were the same, and imported by the company.

RELATION TO OTHER KNOWN DISEASES.

In the course of my visit I found, as had been stated, that a considerable proportion were attacked by Beri-Beri. Unfortunately, few of the cases were uncomplicated. In every fatal case there was acute dysentery and recent or acute malaria, and a large number of the other cases either had dysentery, malaria, or anchylostomiasis. These diseases were prevalent in this mining district. In persons who had not got Beri-Beri, 51 out of 170 were markedly anæmic. An examination was made of as large a proportion of those at work as was found possible, with a view to ascertaining the presence or absence of a prodromal stage. The diseases already mentioned, though frequent accompaniments and sometimes precursors of Beri-Beri, are in no sense the initial stage of the disease, though they may precipitate its onset.

In the epidemic in the Kwala Lumpur Gaol, in 1901—1902, there was little dysentery, no malaria, and though doubtless some of the patients harboured anchylostomes in small numbers, no general anæmia from anchylostomiasis.

In Singapore, in the outbreak in 1903, there was a good deal of dysentery in the gaol, but no malaria. In the admissions to the Kwala Lumpur District Hospital some of the patients had had a recent malaria, and some of the deaths, attributed to malaria, were really due to malaria combined with Beri-Beri, a fatal combination, but many of the patients had had no symptoms of malaria, and at a post-mortem examination, no traces of

malarial pigment, or other evidence of recent malaria was found. Similarly, it can be shown that tuberculosis has no part in the production of the disease, as it is very rare at Sungei Lembing, though not uncommonly found associated with Beri-Beri in Kwala Lumpur. These diseases, as complications, greatly increase the mortality from the disease, but are not the cause of it. Some of them, *e.g.*, malaria and dysentery, do cause a form of peripheral neuritis, which, in isolated cases, may be mistaken for Beri-Beri.

PERIOD OF EXPOSURE.

In an area where Beri-Beri is endemic, it is difficult to determine the time of exposure required to produce marked signs of the disease. Persons complain of symptoms and show definite signs, only after they have been ill for some days. Various factors have to be considered.

It is no doubt possible that some contract the disease in China or in Hong-Kong whilst awaiting shipment, and it is more than probable that, occasionally, the exposure to infection commences on board, as these ships also carry returned coolies, sometimes Beri-Beri convalescents, back to China.

The length of the voyage to Singapore varies a little, but is usually about a week, and sometimes a day less. There do not appear to be any specially infected ships now on the route, as there is no sufficient difference between the incidence of the disease in those brought by one ship or another.

In Singapore, the disease is endemic, but not to the same extent as in Sungei Lembing. The duration of the stay in Singapore, before the contract is signed, is subject to considerable variation: 86 were only one day in Singapore, and 77 per cent. were a week or less, and nearly 20 per cent. two weeks or less. Of the remainder, 16 were under a month, and only three over a month in Singapore, before they signed their contracts. Of the cases of Beri-Beri, 84 per cent. were in those who had been a week or less in Singapore, including six cases in men who had only been a day; and 13 per cent. amongst those who had been less than two weeks. One case occurred in a man who had been over four weeks in Singapore, but as it did not develop for over six months after his arrival in Sungei Lembing, it is improbable that the time spent in Singapore was responsible for the attack.

On the whole I think we can fairly exclude both the ships and Singapore from playing an *important* part in the production of any but a small proportion of the attacks.

From Singapore, usually the day after signing the contract, the coolies are taken by sea to Kuantan. They may be delayed a day or two at Kuantan, and then go by boat up the river to Sungei Lembing, where they are medically inspected the day after arrival. During this period they are either on a ship which may carry cases of Beri-Beri, in a town where there is a moderate amount of Beri-Beri, or in boats which are used, among other things, for taking Beri-Beri patients from Sungei Lembing to the sea-side. The period from signing the contract to the medical examination is 5 to 8 days, very rarely more, and during the greater part of this time they should be considered as in an endemic area.

At Sungei Lembing they may be employed in the mines pushing trucks, wood cutting, &c. A few are employed in connection with domestic work for Europeans. They may be working directly under European management, or sublet to Chinese contractors. The barracks are good, and kept fairly clean; some of them are decidedly better than in Selangor mines, and in none is there so much overcrowding as in the Selangor mines, or in the more thickly-populated quarters of the towns. Cases of Beri-Beri occur amongst all classes, and no barrack is exempt, though at any one time more cases may be found from one than from any other gang. As the Sinkehs are often changed from one contractor to another, and from one occupation to another, it is impossible to make any statistical comparison.

In all cases the Sinkehs are fed by contract. On the whole they are badly fed, though some of the sub-contractors do their best. Fresh meat is scarce, and no fresh vegetables are grown, or can be purchased. Dried vegetables, dried fish, and rice are the staple articles of diet. The rice is imported by the Company and sold to the contractors at a uniform price of \$5.00 a bag. As this is \$2 or \$3 less than it could be procured for otherwise, it can be assumed to be practically the only rice used. It is a fair quality Rangoon rice.

Malaria, Dysentery and Anchylostomiasis are prevalent diseases, and many of the men are naturally of poor physique, and accustomed only to sedentary occupations.

Beri-Beri soon breaks out amongst the new arrivals; and occasionally two or three days after arrival, so that it is certain these persons must have acquired the disease before arrival, in all probability in Singapore.

For 1904, detailed information as to the date of leaving China, arrival in Singapore, ship, &c., has been kindly supplied to me by the Protector of Chinese for each Sinkeh. The date of contract, and that of Medical Examination, were supplied to me at Sungei Lembing. The date given as that of onset, is that on which the disease was diagnosed by the Medical Officer of the Company or myself. In all, or nearly all, a history of several days' illness previous to this could be obtained, and this history included some of, if not all, the symptoms associated with Beri-Beri, such as cardiac distress, cedema, numbness or weakness of the limbs. The period of residence given as that of exposure before acquiring Beri-Beri is therefore, in reality, longer than would be given if the date of the first definite symptoms was obtainable, as it includes the time between the onset of symptom and the admission of the patient to Hospital. Two cases developed on the second and sixth day after arrival in Sungei Lembing, and had no doubt acquired the disease before arrival. One of these had left Hong-Kong 29 days previously, and arrived in Singapore 15 days before the onset. The third shortest period was 20 days after arrival at Sungei Lembing, 30 days after arrival at Singapore, and 38 after leaving Hong-Kong. There were two other cases after less than 30 days' residence at Sungei Lembing, but in both of them an exceptionally long time, seven days, had been spent at Kuantan. There were five cases after between 30 and 40 days' residence, and seven cases between 40 and 50 days' residence.

In the year 1904, 559 Sinkehs, engaged in Singapore, were brought to the mines and commenced work as follows:—

Jan. 6th, 55; Jan. 22nd, 18; Feb. 5th, 48; April 4th, 9; April 20th,

23; May 1st, 17; June 13th, 40; June 21st, 15; July 10th, 59 and 19; Aug. 1st, 51; Sept. 1st, 45; Oct. 1st, 48; Oct. 16th, 24; Nov. 7th, 30; Nov. 10th, 34; Nov. 27th, 16; Dec. 20th, 8. They were sent by steamer from Singapore to K. Kuantan, and thence by boat to the Sungei Lembing as usual.

My observations were made in the latter half of December and in January, 1905, and the notes previously and subsequently are made from the Hospital and other records of the Pahang Corporation, and from information supplied to me by Dr. Dodds, then Medical Officer to the Company.

Amongst those who arrived on January 6th, the first case of Beri-Beri occurred on the 5th February, 30 days, and the second on the 23rd, 48 days, after arrival; two cases occurred in March and one each in April and May, so that six out of 52 in all were attacked. At the time of my visit, after nearly a year's residence, six of the persons who had not had Beri-Beri were examined, and the knee-jerks were absent in one. Of the 18 who arrived on Jan. 26th, one developed the disease by the 28th. No doubt infection had occurred before arrival. There was one case on 21st March, nearly two months after arrival, and one case on June 11th.

Of the arrivals on Feb. 5th, 48 in number: first case on March 17th, and the second on the 21st, 43 and 47 days after arrival; six cases occurred in April and three in June.

Of the arrivals on April 4th and 10th, 32 in number, the first case occurred on June 6th, after two months' residence; and in August and October each, one case occurred.

On May 1st there were 17 arrivals. Amongst these one case occurred in August, and two in October.

In June there were two batches, one of 40 on the 9th, and another of 15 on the 25th. The first case occurred on August 3rd, 54 days after arrival; there were two cases in September, four in October, and three in November.

On July 10th 78 Sinkahs arrived. The first case occurred on Sept. 19th, 70 days after arrival; one case in October, another in November, and four in December.

Fifty-one arrived on July 27th. One had the disease on arrival; the next cases were on the 16th of September, 50 days after arrival, and another case occurred on the 26th, 60 days after arrival. There were two cases in October, three in November, and none in December.

On September 1st there were 45 arrivals. The first case occurred on the 21st, three weeks after arrival. On October 5th there were two cases, 35 days after arrival; another on 16th, 46 days after arrival; another on the 24th, 54 days after arrival. There were four cases in November and six in December.

On September 30th there were 48 arrivals, and on October 14th 24 more. Of the arrivals on September 30th, the first case diagnosed was on November 22nd, after 53 days' residence, and four cases occurred in December. Of the arrivals in October, the first case was on Nov. 11th, after 29 days' residence,

and two cases occurred in December. No cases occurred amongst the 80 persons who arrived in November till January, but seven cases occurred amongst them in that month, six in February, and three in March.

Of the 21 arrivals in December, three were attacked towards the end of January, and two in February.

On December 31st, 1904, there had been in all 559 Sinkehs, and of these, in two the disease had occurred on or before arrival at the mines. Among the remaining 557 persons, no cases occurred during the first three weeks, but one occurred on the twenty-second day and two between the fourth and fifth weeks, three in the sixth week, two each in the seventh and eighth weeks, and four in the ninth week.

The largest number of cases, 33 and 34, were in the third and fourth months, and of the 559 persons, 439, not deducting deaths and absconders, had completed that period at the date of observation, so that in the third month 5.2% were attacked. Excluding deaths and absconders, there were only 185 who commenced the second half-year, but of these only one was attacked, and in the last quarter of the year, none of the remaining 50 were attacked, though it is the most unhealthy period. It is noteworthy that during the period when the number of cases, Sinkehs and coolies, were at their highest, and therefore infection most probable, or the circumstances causing the disease most prevalent, *i.e.*, January, February, November and December, five out of the six cases which developed after less than six weeks' residence occurred.

The larger figures, obtained by grouping for a series of years all the cases that occurred according to the interval between the date of detection of the disease and that of the signature of the agreements, in Singapore one to two weeks before arrival in the district, show a similar variation in the incidence with length of residence.

These larger figures, however, show further that there is a tendency to an increase in the number of cases in November, December, January, and February, even in those longer resident. When these months coincide with third and fourth months of residence, a far larger proportion are attacked than when the Sinkehs are introduced at such a season that this dangerous period of residence is passed in a more healthy period of the year. Thus of 560 Sinkehs imported in March, April, and May, 108 or 19.3 per cent. were attacked in the course of the period of one year, whilst of 594 introduced in October, November, and December, 187 or 31.4 per cent. were attacked. The difference is sufficiently marked to render it advisable that such immigrants should, as far as possible, be introduced in the first half of the year, and that in the last quarter such introduction should be avoided.

Of the years considered 1898-1904, from May, 1901, to April, 1902, both inclusive, only 47 Sinkehs were introduced, and 28 of these were introduced between August and December. Of these, seven acquired Beri-Beri, one in the second month's residence, two each in the third and fourth, one in the fifth, and one in the eighth, about the ordinary proportions for the early months; but this was accompanied and followed by a great decrease in the amount of Beri-Beri, and when the importation of Sinkehs was renewed, from April to July, 1902, 255 Sinkehs were introduced, and the rate at which these new immigrants were affected was much reduced, only eight, including a

case probably infected before arrival, developed the disease in the first six months, or 3.1 per cent. Cases become common in December, January, and February, and amongst the cases imported later, the incidence again becomes great.

Beri-Beri cases in Sinkehs, imported 1904, Sungei Lembing, up to March 30th, 1905.

| Months | | 1 | | | | 2 | | | | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|-----------|-----|--------|---|---|---|--------|---|---|---|----|----|----|---|---|---|---|----|----|----|
| | | Weeks. | | | | Weeks. | | | | | | | | | | | | | |
| Date. | No. | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | | | | | | | | | | |
| Jan. 6 | 55 | | | | 1 | 1 | | | | 2 | 1 | 1 | | | | | | | |
| " 22 | 18 | 1 | | | | | | 1 | | | | 1 | | | | | | | |
| Feb. 3 | 48 | | | | | 1 | 1 | | | 6 | | 3 | | | | | | | |
| April 6 | 8 | | | | | | | | | 1 | | 1 | | 1 | | | 1 | | |
| " 26 | 32 | | | | | | | | | | | | | | | | | | |
| May 1 | 17 | | | | | | | | | | 1 | | 2 | | | | | | |
| June 9 | 55 | | | | | | | | 1 | 2 | 4 | 3 | | 1 | | | | | |
| " 25 | 15 | | | | | | | | | | | | | | | | | | |
| July 9 | 39 | | | | | | | | | 1 | 1 | 1 | | 2 | 2 | 2 | | | |
| " 27 | 51 | 1 | | | | | | 2 | | 2 | 4 | | 1 | 3 | 1 | | | | |
| Sept. 1 | 45 | | 1 | | | 2 | 1 | | | 4 | 6 | 6 | 2 | | | | | | |
| " 30 | 23 | | | | | | 1 | | | 4 | 8 | 1 | 2 | | | | | | |
| Oct. 14 | 24 | | | | 1 | | | | | 2 | | 1 | 1 | | | | | | |
| " 25 | 31 | | | | | | | | | 2 | 3 | 2 | | | | | | | |
| Nov. 11 | 34 | | | | | | | | | 5 | 4 | 1 | | | | | | | |
| " 25 | 16 | | | | | | | | | 2 | 2 | 3 | | | | | | | |
| TOTAL | 551 | 2 | 1 | 1 | 2 | 3 | 2 | 2 | 4 | 33 | 34 | 24 | 8 | 7 | 3 | 2 | 1 | | |
| In months | | 5 | | | | 11 | | | | 33 | 34 | 24 | 8 | 7 | 3 | 2 | 1 | | |

The general principle that at Sungei Lembing the onset of Beri-Beri occurs after a residence of three or four months, is shown by the figures abstracted from the Hospital records for the last six years, comprising 497 cases out of 1,942 Sinkehs. Of these cases the numbers that occurred in the different months of residence were as follows :-

| Months— | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---------|----|----|----|----|----|----|----|----|----|----|----|----|
| | 10 | 48 | 91 | 82 | 72 | 46 | 34 | 29 | 30 | 21 | 20 | 14 |

Two hundred and forty-five, nearly half the cases, occurred in the third, fourth and fifth months after the date of contract, and therefore after one or two weeks shorter residence in Sungei Lembing. The figures from which these tables have been compiled appear to have been carefully kept, but it must be remembered that the time taken is the date of admission to hospital, and that most of the patients have been ill for some days before admission, and therefore that the period of exposure required is slightly less than these figures indicate.

This period of about three months may therefore be taken as the common period of exposure to the conditions of life at Sungei Lembing before the patient is sufficiently ill with Beri-Beri to be admitted to hospital; but it must be added that not only do some cases acquire the disease in a

shorter time, but that in others a longer exposure is required. Even amongst the Sinkhehs, who having fulfilled their agreements (300 working days—practically a year), and remain at the mines, cases occur from time to time among those who escaped in the first year's residence.

It follows, therefore, that the chances of infection vary :—

- (a) Either on account of unequal susceptibility or varying power of resistance.
- (b) Or that the exposure to risk of infection varies in different individuals.

As in most of these questions, that of relative immunity is one of difficulty. It is, however, clear that, as so large a number do not get the disease in the ordinary period, and do subsequently, the immunity, if it exists, is a temporary one and liable to be lost.

The inequality in the incidence is opposed to any of the views that the disease is caused by the consumption of a special article of diet, as the food for each batch of Sinkhehs is the same and supplied by the same contractor.

A closer examination of the figures since 1899, by dividing them into quarters, shows that from the second quarter of 1901 to the first quarter of 1902, there were exceptionally few cases, only nine in all; none in the first, one in the second, five in the third, and one each in the fourth, fifth, and twelfth month, *i.e.*, such cases as there were, occurred after the usual period of residence. In the next quarter there was only one case in the first six months, but eight in the second half-year; and in the next year, in each quarter, the incidence was great, but though there was a tendency to early development of a few cases, the great mass of the cases occurred amongst men in their second half-year of residence.

The number of cases has varied, and according to the varying number of cases, the whole period can be divided into four. The first was from June 16th, 1899, to March 31st, 1901.

In this period the cases occurred within the periods after signing the contracts in Singapore, as here shown.

| Months— | 1 | 2 | 3 | 4 | 5 | 6 | 7-12 |
|---------|---|----|----|----|----|----|------|
| | 7 | 37 | 56 | 54 | 43 | 21 | 50 |

or out of 268, 110 or 41 per cent. of the cases were contracted in the third and fourth months after signature, or after $2\frac{1}{2}$ to $3\frac{1}{2}$ months' residence, and only 50 or 19 per cent. in the last six months, or at the rate of 6.6 per cent. for a corresponding period of two months.

In the second period, from the second quarter 1901 to the first quarter of 1902, both inclusive, there were very few cases. These occurred at the following periods after signature of the contracts.

| Months— | 1 | 2 | 3 | 4 | 5 | 6 | 7-12 |
|---------|---|---|---|---|---|---|------|
| ... | 1 | 1 | 5 | 1 | 1 | 1 | 1 |

ten cases in all; and half of these in the third month. In the third period, following immediately on the second one, the cases again were numerous: but there was a complete difference in the periods required for the development of the disease, from that met with in the first two periods—from June, 1899, to March, 1902.

In this third period, from the second quarter 1902 to the second quarter of 1903, both inclusive, the incidence was

| | | | | | | | |
|---------|---|---|----|----|----|----|------|
| Months— | 1 | 2 | 3 | 4 | 5 | 6 | 7-12 |
| | 2 | 4 | 13 | 14 | 22 | 17 | 87 |

Out of 159 only 27, or under 17 per cent. of the cases acquired the disease in the third and fourth months, as compared with 41 and 50 in the two previous periods; and 54 per cent. of the cases were acquired in the second half-year of residence, as compared to 19 per cent. and 10 per cent. respectively in the two preceding periods.

Subsequently to this, the third period, the incidence was the usual one to the end of the year.

| | | | | | | | |
|---------|---|---|----|----|---|---|------|
| Months— | 1 | 2 | 3 | 4 | 5 | 6 | 7-12 |
| | 1 | 8 | 16 | 12 | 9 | 5 | 3 |

Out of 54 cases 28, or 51 per cent. occurred in the third and fourth months, and only three, or under 6 per cent., in the second half-year.

The figures for 1904 have been given in more detail, and as in this period and in the first period, from 1899 to March, 1901, show the incidence to be the greatest in the third and fourth months.

It will be obvious that the two exceptional periods are those called the second and third.

The explanation of the peculiarly small number of cases in the second period is simple, as, whilst in the previous and succeeding years 500 to 600 men were imported each year, in that period only 61 were imported between April 1st, 1901, and March 30th, 1902.

In the third period, a year and a quarter, 680 Sinkehs were imported; but this, though it explains the increased number of cases, does not in itself explain why so large a proportion of the cases amongst these occurred after prolonged residence instead of, as before and after, in the early months of residence. The exact table of successive importations shows that the change back to the normal condition was a gradual one.

| Months of Residence. | No. of Persons. | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|----------------------|-----------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| 1902. | | | | | | | | | | | | | |
| April ... | 23 | ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... |
| May ... | 74 | ... | ... | ... | ... | ... | ... | ... | ... | 1 | 1 | 2 | 2 |
| June ... | 46 | 1 | ... | 2 | ... | ... | 1 | ... | ... | 1 | 1 | 2 | 1 |
| July ... | 51 | ... | 1 | ... | ... | 1 | 1 | ... | ... | 2 | 1 | ... | ... |
| August ... | 61 | ... | ... | 1 | 3 | 4 | 3 | 1 | 1 | 1 | 1 | ... | 1 |
| September ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| October ... | 124 | ... | ... | 1 | 3 | 1 | 1 | ... | ... | 1 | ... | 1 | 2 |
| November ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... |
| December ... | 15 | ... | ... | 1 | ... | ... | 2 | ... | ... | ... | ... | 1 | ... |
| 1903. | | | | | | | | | | | | | |
| January ... | 18 | ... | ... | ... | 1 | 2 | 1 | ... | ... | ... | 1 | 1 | ... |
| February ... | 18 | ... | ... | ... | ... | 1 | 1 | 1 | ... | ... | ... | 2 | ... |
| March ... | 70 | ... | ... | 3 | 1 | 5 | 1 | 1 | ... | 3 | 6 | 1 | 1 |
| April ... | 50 | ... | ... | 2 | 1 | 1 | ... | ... | 6 | 4 | 2 | 1 | 1 |
| May ... | 34 | ... | ... | 2 | ... | ... | 2 | 3 | 4 | 3 | 1 | 1 | 1 |
| June ... | 96 | 1 | 1 | 1 | 5 | 3 | 7 | 10 | 4 | ... | 1 | 2 | 1 |
| TOTAL ... | 680 | 2 | 2 | 11 | 16 | 18 | 20 | 16 | 15 | 15 | 16 | 14 | 10 |

During the same period the number of free coolies, *i.e.*, those over one year in the country, admitted to the hospital with Beri-Beri, was only 11, and for six months of the period, June to November, there were no cases among them. The average yearly number of coolies, so admitted, is over 30.

This late incidence of the disease, following a period when so few people were imported, is significant. The occasional occurrence of cases all through the period showed that the conditions necessary for the development of the disease were present, but instead of, as commonly, requiring an exposure of three months, an exposure of over six months was necessary for the same proportion of persons to be attacked.

It became an instance of importation of susceptible people into a district where only a few persons with the disease were present, and where, consequently, infection was late, instead of, as before and since that time, of the importation of susceptible persons into a district where cases were numerous and infection rapid.

The "Endemic index" on that explanation would therefore appear to depend on the number of cases of Beri-Beri in the district at the time of the introduction of the susceptible newcomers.

The varying incidence of the disease at Sungei Lembing is therefore explicable by the hypothesis that Beri-Beri is introduced to a group or class by man, and spreads in some manner from man to man, *i.e.*, that it is an infectious disease.

From the nature of the lesions which cause the characteristic group of symptoms, these lesions are secondary, and therefore the primary cause must be the one which is transmissible from man, but not necessarily directly to man, as the mode of conveyance may be indirect.

Other hypotheses.—Before finally adopting this conclusion as a basis for further enquiry, it is well to consider the other hypotheses advanced, *viz.* : That Beri-Beri is secondary to the absorption by man of a poison originating or existing outside the human body. One of these, "earth," "ground" or "house" infection, assumes that the poison of Beri-Beri is exhaled or emanates from the ground. If it is further assumed that this poison originally is derived from a person infected with Beri-Beri, it differs from the theory of infection of man by man only in stipulating for a prolonged life for the producer of the poison, and indefinite power of multiplication outside the human host. It may at once be admitted that cases do occur, as in the instance of house infection quoted, in which, after the removal of a person with the disease, other persons inhabiting the house shortly after acquire the disease, but this is no more than may happen with undoubtedly infectious diseases carried by intermediate hosts, such as Malaria and Yellow Fever.

Earth, ground, or place infection has had many advocates. The repeated recurrences of the disease in ships, prisons, and institutions has been urged in support of it.

The abrupt cessation of some of the outbreaks, as of that in January, 1902, in Kwalla Lumpur Prison, and in October, 1904, in Singapore Gaol, both institutions covering several acres of land, is opposed to any views which involves a belief in the prolonged existence of the cause of the disease outside a human host, or in the indefinite reproduction of this cause in earth or

buildings, as in neither case were there any peculiar meteorological conditions, any general disinfection or other change which could possibly simultaneously affect so large an area, and in cells so differently circumstanced, as those in the upper and lower tiers of the gaol.

Attempts at disinfection of places have not been very successful. Most of these, however, have been associated with constant introduction of fresh cases of the disease.

A favourite argument for place infection is the alleged effect of removal from an infected locality. It is stated that persons removed from such a locality recover more rapidly, and that the death rate is much smaller. The locality to which such patients are removed is usually a healthier one, and frequently better or more varied food is available. Of the instances in Kwala Lumpur, the good effect of the removal of prisoners from Pudu to the old gaol in 1895-1896, was stated by both Drs. McClosky and Travers to be most marked. The removal from a damp prison, with new cement floors and walls, to an old building with wooden floors, may have been the reason for the better results.

Further, the removal was from a place where there was a constant influx from outside of cases in the early stage of the disease to a place where no such influx was taking place. At Sungei Lembing it is customary to transfer Beri-Beri cases from the hospital to a hospital near the mouth of the Kuantan River.

This hospital is built on piles over the water, earth is only exposed under the buildings during the low tides. There is little difference in the mortality in the two hospitals, although at the Kuantan Hospital fresh fish and fresh vegetables are obtained, and rarely at the Sungei Lembing Hospital.

The death rate from Beri-Beri at Jugra is very high, though the disease is rare in the neighbourhood, and that at Port Dickson is low, whilst there is no endemic Beri-Beri in that place. These divergencies are, as Durham has pointed out, no more than occur in the various hospitals in Selangor. In some places, as at Kajang, the mortality is persistently low; in others, as Kuala Kubu, persistently high.

Further, in all questions of transfer of patients, a certain amount of selection consciously or unconsciously is made. The severest cases, those rapidly fatal, in which the heart is early and severely affected, are unsuitable for transfer, whilst the subacute cases and those with little or no cardiac implication, are transferred, and amongst these the death rate is, under any circumstances, comparatively low. As was pointed out to me at the Seamen's Hospital in the Albert Docks, by Mr. Conynghame, who had had considerable experience in British Borneo, the class of cases that we received from the ships were similar to those which recovered in Borneo, not to those who died; and that, therefore, with naturally selected cases such as these, a low mortality was to be expected.

The recurrences in places or in ships are often at prolonged intervals, and to assume that the poison is formed in and derived from the ground or structures, requires the further assumption that the poison-forming agent may remain latent, and in the gaols at least, be aroused to renewed activity without any meteorological or other change in conditions at irregular and uncertain intervals.

The freedom of prison warders of all nationalities from the disease, though they must be exposed to the ground emanations nearly to the same extent as the prisoners, and the freedom of Europeans who are exposed to the same extent, as other prisoners, is not explicable on this hypothesis.

The varying conditions of ground in places where Beri-Beri is rife is well shown by Durham to be directly contrary to this view. In the well ventilated lines with raised floors in Christmas Island, the disease was more prevalent than in the moderately ventilated prison cells in some gaols, or in ill-ventilated crowded forecastles occupied by the crews of ships.

A second division is the hypothesis of chronic poisoning by a substance either of known definite composition, or of unknown or uncertain composition, the product of certain forms of destruction of food stuffs.

Arsenic and other mineral substances, as is well known, produce nerve lesions which, in some respects, closely resemble those of Beri-Beri.

In the Federated Malay States, arsenic occurs in nature with the tin to a large extent, and is very widely diffused. The possibility of arsenical poisoning occurring must therefore be admitted, particularly in the mines or where smelting is carried on.

It cannot however be admitted as the cause of Beri-Beri, as even in the mines such as Sungei Besi, where arsenic is common, the Tamils escape and the Chinese in adjoining mines are attacked. In the agricultural districts, such as Krian, where there is no arsenic or arsenic containing ore dealt with, the Chinese get Beri-Beri, and the Tamils escape.

Further, in the prisons no work at all remotely connected with arsenic is carried on, and yet when the disease is epidemic, a larger proportion of the prisoners are attacked by Beri-Beri than in the most arsenical mines.

Tobacco, as prepared for the Chinese, is said to be sometimes contaminated with arsenic, and also in the comparatively small amount of beer consumed, and other drinks, arsenic might be present; but none of these articles are allowed in the prisons where Beri-Beri has so frequently occurred.

Dr. T. S. Kerr had every article of food in use in the Singapore Gaol analysed by Mr. Burgess, the Government Analyst, and no arsenic was found in any of the foodstuffs, nor is arsenic in any form used in the preparation of any of the foodstuffs, rice, curry stuff, meat, salt fish, &c., used in the gaols.

Arsenic has been found in the hair of persons with Beri-Beri. None of the ordinary symptoms of arsenical poisoning are present either before or during an attack of Beri-Beri. This is most unusual in cases of arsenical neuritis.

Pigmentation of the skin, and particularly of the parts of the legs exposed to sunlight, is sometimes present in very chronic cases of Beri-Beri; but this is no more than might be expected from the nerve and vascular changes in these parts.

Herpes Zoster I have only once seen in Beri-Beri, and Durham records a similar experience. The skin lesions so frequently resulting from arsenical poisoning are conspicuous by their absence in Beri-Beri.

Alcohol causes a neuritis not unlike that of Beri-Beri, but few of the Chinese consume alcohol in any form, and still fewer to excess; further, none is allowed in the prison. Even if it were assumed that decomposed rice contained a sufficient amount, the thorough boiling of the rice would expel most of it. As a matter of fact, alcohol is very rarely present, even in minute amounts, in decomposed rice. Acetone is common.

Oxalic acid has been suggested as a possible cause. Small quantities are found in certain forms of fermentation of rice, but only exceptionally, and these forms of decomposition are more extreme than anything which could possibly occur with the inspection of the food that is practised in the gaols.

Many of the theories that have from time to time been held by divers observers, have attributed the disease to diet or to some articles of diet which contain an unknown poison. Of these theories that of *Nitrogen Starvation* or too large a proportion of carbohydrate food, particularly of rice, has attracted a great deal of attention, as the experience in the Japanese Navy, formerly decimated by Beri-Beri, was that an increase in the nitrogenous food was followed by a great reduction in the amount of Beri-Beri. This improvement in diet was one of the many changes made in that Navy, and experience has shown that with a diet as physiologically correct as that in the Japanese Navy, Beri-Beri may still be rampant. Dr. Hamilton Wright's experiment included a period of eight months, during which the nitrogenous food was in excess of that in the Japanese Navy; there were 106 cases out of an average strength of about 500. Further, the food in the Selangor Mines, where the coolies buy their own food, is good, and there is no deficiency of any of the main constituents necessary for a diet, not only physiologically sufficient, but even super sufficient, as regards nitrogenous food.

In other instances, as in that observed by Durham at Christmas Island when, during a period of some months, owing to a failure in the communications, the supply of food, and particularly of nitrogenous food, was decidedly below the proper or usual standard, there was less Beri-Beri than usual.

In the prisons, the periods when Beri-Beri was rife and when the disease was absent, occur without any change in the relative or absolute amounts of proteid and carbohydrate food, or in the nature of the food or the cooking. During these periods the class of work and hours of work have not been changed, so that there was no such change in the conditions as would render a diet, sufficient during one period, insufficient in another on account of a difference in occupation. As an instance the experience of Kwala Lumpur Gaol, 1902, 1903 and 1904, may be given, as in these years the number of cases of Beri-Beri was respectively 891, 55 and 26, and in 1903 for no less than seven months there were no cases at all.

Insufficient food, a diet physiologically unsound, or even decayed food, are common enough in countries with climates not unlike that of the Malay Peninsula, as well as in other countries, and yet no Beri-Beri results.

We can therefore, in my opinion, definitely exclude insufficient or badly-designed diets as direct causative factors in the etiology of Beri-Beri.

Like most persons with a practical experience of the disease, I am, however, strongly in favour of a liberal dietary as to some extent a preventative

of Beri-Beri. An improvement in diet, and particularly an increase in the amount of fresh vegetable and animal food is of great value in diminishing the spread of outbreaks. An improvement in diet, if it be insufficient, is one of the essentials in dealing with a place where the disease is endemic, in association with measures directly undertaken to prevent the further introduction or spread of the disease.

This view is in practice adopted by all as a part of the routine procedure in any organised attempt to reduce the amount of this disease. The success of these organised attempts, as in the instance of the Japanese Navy, is often attributed to this part of the scheme alone.

Much more importance must be attached to theories based on the assumption that the poison of Beri-Beri is formed in certain articles, or in articles of food prepared in certain ways, and that the poison thus taken with the food, when absorbed, causes as a secondary result the nerve changes.

The experience of the gaols is of value in considering this theory. There is no chance, in the larger prisons, of supplementing the diets, and consequently, all the substances used as food or in the preparation of food are known; if the theory be correct, some of these foodstuffs must be implicated.

The basis of all diets in the prisons in the Malay States is rice. Other articles include salt fish, fresh meat, varying kinds of dried vegetable food, and occasionally fresh vegetable food. Dietary of various gaols are appended. It will be seen that the two classes of food that must be mainly responsible are salt fish and rice.

Fish can, I think, be excluded, as during Wright's experiment in Kwala Lumpur Gaol for eight months, no fish at all was given, meat only, and yet during this period there were 106 cases of Beri-Beri.

Moreover, salt fish is largely consumed in the West Indies and in Demerara, where Beri-Beri either does not occur or is extremely rare. I saw no cases of the disease in a hospital, with a daily average number of inmates of over 700, in the course of six years.

Rice is the article of diet which many observers—who hold the view that the disease is due to certain qualities of food under certain circumstances—consider of most importance.

In considering this question in all its bearings, it is essential to know the length of exposure to influences producing Beri-Beri in a place where the disease is prevalent. It will be seen from the Sungei Lembing figures, that the usual period of exposure there was 3, 4, or 5 months, with the exception of a time during which the prevalence of the disease was less than usual, when the period of exposure was more prolonged.

The figures compiled by Travers during the outbreak, 1900-1903, for the Kwala Lumpur Gaol, for 256 consecutive cases with no previous history of Beri-Beri, are as follows :—

| Period of time between date of sentence
and date of appearance of symptoms of
Beri-Beri. | | | | No. of Prisoners
contracting
Beri-Beri. |
|--|-----|-----|-----|---|
| Under 1 month | ... | ... | ... | 11 |
| Between 1 and 2 months | ... | ... | ... | 45 |
| 2 and 3 | " | ... | ... | 67 |
| 3 and 4 | " | ... | ... | 40 |
| 4 and 5 | " | ... | ... | 27 |
| 5 and 6 | " | ... | ... | 25 |
| 7 and 12 | " | ... | ... | 8 |
| Over 1 year | ... | ... | ... | 13 |

A large number of the prisoners are short-sentence prisoners and the numbers under six months are naturally much greater than those over six months. Admitting that these figures do not represent correctly the relation between length of residence in gaol and the relative number of cases, they do at least prove that a large number, 152, contracted the disease after exposure to the gaol conditions from one to four months. The figures taken with those from Sungei Lembing are proofs that a comparatively short exposure, under four months, is sufficient for the development of marked signs of the disease, *i.e.*, the nerves are affected.

I use the term "period of exposure": An incubation period would necessarily be shorter in most cases unless the persons were infected at the commencement of this period. From the outbreak in Penang the "incubation period" may be as short as 10 to 20 days. Hamilton Wright considers that it may be as short as 10 to 12 days.

Durham, at Christmas Island, examined "a number of Beri-Berics who certainly had not been more than three months on the Island." He examined one batch of men who had been sixteen days on the Island, and, with the exception of two with slightly increased knee-jerk, he found them to be healthy. On the 66th day one of them died of Beri-Beri, and twelve days later, out of nine of the gang, only two could be passed as free from suspicion of Beri-Beri. These men were not Sinkeh's.

The District Officer and Dr. Giddy furnished Durham with figures showing the length of residence of 100 consecutive fatal cases of Beri-Beri on the Island.

| Length of Residence. | | | | No. of Deaths. | |
|----------------------|-----|-----|-----|----------------|----|
| 2 months | ... | ... | ... | 11 | 32 |
| 3 " | ... | ... | ... | 21 | |
| 4 " | ... | ... | ... | 6 | |
| 5 " | ... | ... | ... | 12 | 25 |
| 6 " | ... | ... | ... | 7 | |
| 7 " | ... | ... | ... | 4 | 9 |
| 8 " | ... | ... | ... | 2 | |
| 9 " | ... | ... | ... | 3 | |
| 10 " | ... | ... | ... | 2 | 12 |
| 11 " | ... | ... | ... | 8 | |
| 12 " | ... | ... | ... | 2 | |
| 1—1½ years | ... | ... | ... | 20 | |
| 1½—2 years | ... | ... | ... | 1 | |
| Over 2 years | ... | ... | ... | 1 | |

The contract time for Sinkehs was one year, and few were sent back to Singapore before their contracts mature. No doubt many of these patients had been ill for a month or more before death, and in any case, the periods given will be in excess of those required for the disease to be acquired.

RICE CONSIDERED AS AN OCCASIONAL CAUSATIVE AGENT IN THE PRODUCTION OF BERI-BERI.

There are many advocates of the supposed connection between the consumption of rice and the occurrence of Beri-Beri. The reports of some of these contain statements that are not quite correct, and, unfortunately, other observers have attached so much importance to these defects that the main question has sometimes been lost sight of.

A strong *primâ facie* case for certain rices as a causative agent has been made out by several men of experience. One of the more recent articles is one by Braddon, who has for many years consistently upheld, with slight modifications, the view that rice parboiled before husking, rarely contains the "poison" of Beri-Beri, whilst, when not so treated, it sometimes does, and always may have, this poison. In this article I make considerable use of the arguments advanced by Braddon.

All races resident in the Malay Peninsula consume a certain amount of rice, and it is the staple carbohydrate food of Chinese, Tamils and Malays.

The Malays in the police, or in other forms of Government service, those in private service, and generally speaking, those employed in towns, use rice which has been husked in bulk, and imported into the country as rice. The variety used is that known as Siamese No. 2, but it comes from various parts of the Peninsula, including Province Wellesley. Such rice, husked or hulled some time before use, is called by Dr. Braddon "stale" and "uncured."

The Chinese use either the same quality of rice, Siamese No. 2, or a different variety prepared in the same manner in Burmah, and known as "Rangoon" rice. This Rangoon rice is the rice provided in most of the public institutions. It also is "stale" and "uncured" in Dr. Braddon's nomenclature.

Siamese No. 1 and Rangoon No. 1 are prepared in a similar manner, but are larger grained and sweeter. They are not in use by the poorer classes, but by Europeans, Chinese Towkays, &c. These rices also are included in Dr. Braddon's "stale and uncured" rices, but would only be bought and used when fairly fresh and in sound condition.

The other rices used are called by Dr. Braddon "cured," because the padi is boiled till the husk splits, and then air-dried for a few days before husking.

The best quality is prepared in India, and is known as "Bengal" rice. It is used by such Tamils as can afford it, but not by the coolie class, as it costs twice as much as a similar rice prepared in the Malay Peninsula or in the Straits Settlements. This rice, known as Kedah, Penang or Province rice, is used extensively by the Tamils, though some of these who have been long resident in the country, and most of the second generation, prefer the

Siamese rice. Some of the worst specimens of rice, the oldest, most broken, and most infested by beetles and lepidopterous larvæ, are of this locally cured rice.

The classes using "uncured," "stale" rice are the working classes of Chinese and the inmates of various institutions, and it is amongst these that Beri-Beri is most prevalent, particularly when the diet is not in other respects superabundant.

The Malays using "uncured," freshly-husked rice probably are unaffected, and certainly few if any get Beri-Beri, whilst cases are not uncommon amongst those who use "uncured" rice which has been kept after husking.

The poorer class of Tamils, who are the worst paid members of the community, use "cured" rice, often of very inferior quality, and very rarely get the disease.

Dr. Braddon, in his thesis on the question of the relationship of differently prepared rice and Beri-Beri, divides the rices into two classes, which he terms "uncured" and "cured." The uncured he further divides into "stale" and "fresh."

The use of these terms is not, in my opinion, advisable, as it to some extent prejudices the issue.

Any one of these rices may have been kept for prolonged periods, and much of the so-called fresh rice is much older than some of the stale.

The natives on the small holdings who cultivate rice for their own use, store it in the husk as padi, and husk by pounding immediately or shortly before use. Such freshly-husked rice, however old it may be, Dr. Braddon calls "fresh" rice.

The natives of India and Ceylon keep the rice in husk for a variable period, and boil before husking. The length of time for which the rice has to be boiled varies, but it is continued till the husk splits, and as half an hour or more is required for this, it is certain that most spores as well as organisms in the husk or grain will be destroyed.

The padi is air dried, and the husk removed by milling. Rice so prepared is called by Dr. Braddon "cured." Rice husked by milling without preliminary boiling is called by Dr. Braddon "uncured," and unless the husking is done immediately before use, he calls the rice so prepared "stale."

A difference between the husked and unhusked rice of great importance is that the husking kills the seed, and therefore renders it more liable to be attacked by fungi, &c., than the live seeds. Dr. Braddon lays stress on the physical injury to the seeds, and the possibility of any spores, &c., contained in the husk thereby being able to attack the seeds after husking, when without such injuries entrance would be impossible. The effect of boiling previous to the husking, would necessarily be to destroy the spores in the husk, and remove this possibility of contamination at the subsequent milling.

It will be at once noted that the classes using the "cured" rice are those practically free from Beri-Beri, whilst those using the "uncured" rice include those classes which suffer most from Beri-Beri.

It is not a matter of surprise therefore, that much attention has been given to the question of the possibility of rice being an important factor in the causation of Beri-Beri, and it is one which requires very careful consideration.

The "cured" rices can be excluded from consideration. In the gaols in the greater part of India, where cured rice alone is used, Beri-Beri, though it does occur in various places, is rare as compared with the Federated Malay States.

According to the Returns in the Annual Reports of the Sanitary Commissioner with the Government of India, for 1901 and 1902 and 1903, there were respectively, 108, 84 and 122 cases of Beri-Beri, in the native army. The army strength was 122, 806 to 124, 231. In the gaol population of 117, 203 and 114, 334, there were in 1901 and 1902 nine cases of the disease, and six in 1903.

In 1901 all the cases in the native army were in the Madras Command, and 49 of these in Burmah. No less than 48 in two separate outbreaks were in the 8th Madras Infantry stationed at Rangoon.

In 1902, 83 out of the 84 were among the Madras regiments. The highest number of cases, 34, were amongst the 3rd and 13th Madras Infantry stationed at Singapore.

In 1903, four cases were in troops stationed at Singapore, and 50 more in troops stationed both at Singapore and at Viziangram.

Of the gaol cases in 1901, five were from Burmah, two Madras, and one each Assam and Bengal.

In 1902, five were from Burmah and four Madras.

There is in certain sea-ports, as in Calcutta, a certain amount of Beri-Beri, both amongst Chinese and other races.

Amongst the European troops there were in these three years, 26, 39 and 28 cases respectively. In proportion to the average strength, the rates are as follows :—

| | | | European Troops. | Native Troops. | Gaol Population. |
|---------|-----|-----|------------------|----------------|------------------|
| 1901 | ... | ... | 1 in 2172 | 1 in 1173 | 1 in 13022 |
| 1902 | ... | ... | 1 in 1552 | 1 in 1418 | 1 in 12231 |
| 1903 | ... | ... | 1 in 2683 | 1 in 1022 | 1 in 16821 |
| AVERAGE | | | 1 in 2102 | 1 in 1204 | 1 in 14024 |

The incidence of the disease therefore is least in the prison population and greatest in the native troops, but in this connection it is noteworthy that out of the 314 cases amongst the native troops, 82 were amongst those stationed in an endemic area, Singapore, and 50 were during part of the time at the same station. Excluding these 132 cases, the incidence on the native troops is about one in 5000, or less than amongst the European troops.

This incidence does not support the views of those who hold that rice is the main or sole factor in the causation of Beri-Beri. The Europeans were mainly stationed in Burmah, none from Singapore, and though, no doubt, in some form rice was taken by all or most of them, it would certainly not amount to the 24-32 ozs. per diem supplied to rice-eating native troops.

Amongst the Tamils in Malaya, who all use cured rice, mainly cured in Penang, Beri-Beri is highly exceptional, both in those employed in agriculture and those working at mines, such as Sungei Besi, &c., though the Chinese working in adjoining mines suffer severely.

It is admitted that the geographical distribution of Beri-Beri as a common disease corresponds closely to that of the use of uncured rice, usually "Rangoon."

In the Malay Peninsula the uncured rices used are those prepared in Rangoon (which is the cheapest) and the so-called Siamese from Siam and other native states and Province Wellesley, of several qualities. In addition, the Malays, who live in kampongs, subsist on locally grown uncured rice.

The Malays living in such kampongs are the only class that do not suffer from Beri-Beri, but it must be remembered that their rice is always freshly husked, and is not accumulated in bulk, or liable to any but aerial contamination, and is husked in small quantities before use.

Of the other varieties of "uncured" rice the second qualities of Siamese and Rangoon rice are used by all the poorer Chinese and in public institutions. Frequently both are used, but Beri-Beri has occurred as epidemics amongst those using "Rangoon" only (*vide* Kwala Lumpur), and also amongst those using "Siamese" only (*vide* Batu Gajah).

If rices are to be implicated, both of these are rices which may contain the germ or poison to which Beri-Beri is due.

The better, or rather wealthier, classes of Chinese escape Beri-Beri, though they also use Siamese rice, either the best or second quality, and so also do the Europeans. But with both these classes there is more variety in the diet—a much larger proportion of animal food is consumed—and other vegetable carbohydrates are used in addition. The diet, in fact, is super-sufficient.

It is, however, clear that the mere consumption of "uncured" rice is not in itself sufficient to induce Beri-Beri, even when the diet is little more than is physiologically necessary.

The interval, seven months, at Kwala Lumpur Gaol, is sufficient to show this, as during these periods "uncured" stale rice only was used and no cases originated in the gaol. In Taiping Gaol, during the periods 1897, 1898, and May, 1903, to Feb., 1904, there were no cases of the disease, though "uncured" stale rice only was used.

It must therefore be admitted that "uncured" rice, if the cause of the disease, is only capable of producing the disease at times. Or, in other words, that much of the "uncured" rice does not contain the necessary ingredients for the production of Beri-Beri. The uncured rice is produced in various countries, and in some of these, as in Rangoon, there is little Beri-Beri in the prisons or amongst troops using local rice, as compared with the

number of cases in the Malay States. The causation of Beri-Beri cannot therefore be attributed to any variety or species of rice grown, or to any disease attacking the rice during the period of growth or harvesting, but must be attributed to subsequent changes in the rice during the period of keeping, storing, transit, or cooking. The last of these can, I think, be excluded, as the consumption of rice, whether boiled or steamed, whether cooked in the large quantities required for use in institutions, or in the small quantities required by fossickers, may be followed by Beri-Beri.

The consumption of rice prepared under such diverse conditions is in some instances followed, and in others not followed, by outbreaks of Beri-Beri.

It is to changes in preparation, transit or storing, that attention has to be paid, and it must further be admitted that the active agent is not destroyed by the most thorough cooking.

The supply of rice to the public institutions is as follows :—

Selangor Gaol—Rangoon rice from 1897.

Taiping Gaol—Rangoon rice from 1895.

Kwala Lipis Gaol—Rangoon rice from 1895.

Pekan Gaol—Siamese rice.

Singapore Gaol—Rangoon rice for Kanji, say six ounces. Siam No. 2 usually for the other food up to Jan. 1st, 1903, and from that date Rangoon rice till Nov. 1st, 1904.

In the mines of the Pahang Corporation Rangoon rice only is supplied to the Sinkehs. In most of the Selangor mines Siamese No. 2 is supplied, in others Rangoon No. 2.

It will be seen that in its wider aspects the proof or disproof of a certain possibly diseased rice causing Beri-Beri is no easy matter.

It is not difficult to show that rice, even when long kept and under circumstances where fungi, &c., have every chance of developing, does not necessarily cause Beri-Beri, but in the modified form, viz., that certain crops of rice prepared and stored under certain conditions cause Beri-Beri, whilst when differently stored or prepared it does not produce this result. is difficult to disprove and equally difficult to prove.

The fluctuations in the amount of Beri-Beri in different years in institutions such as Kwala Lumpur, supplied during the whole period with one class of rice, on this theory is explicable, as it may be urged that the conditions under which this "uncured" rice was stored in the one year were such that the poison of Beri-Beri did not develop in the rice, and that in the other years it did.

The greater incidence in the gaol than in the general population, who also used the same class of rice at the same time, does not disprove the theory, as the general population procure rice from several sources, and though, no doubt, many may have used rice similar to that in the gaol, others did not, and few would have used the same kind of rice, uniformly, or continuously. The oft-quoted instance where at Kwala Lumpur, the Leper Asylum, the Lunatic Asylum, and the Prison were all

supplied from a common stock of rice, loses some of its significance when the effect of prolonged residence under adverse conditions in the Malay States is considered. It has been shown that at Sungei Lembing amongst those who escape an attack of Beri-Beri during the first few months of residence, a comparatively small number are subsequently attacked. It is quite possible that in the wretched circumstances under which lepers and lunatics live outside the asylums, that they have been more frequently exposed to the conditions necessary to induce Beri-Beri, and that those who find their way to the asylums are the residue who are little liable to be attacked. The good physique and comparative youth of the great mass of prisoners indicate previous life under fairly favourable conditions, and would not give any reason for the supposition that any degree of immunity existed amongst them.

In brief, though both bodies of men were supplied with the same rice, the two bodies of men had different antecedents which might influence their relative susceptibilities, and reduce the value of the experiment as to the "controls." A more liberal dietary in other respects, and greater variation in it, as well as less confinement and less work are also points which diminish the value of the "controls."

There have been from time to time certain curious coincidences in the increase and decrease of Beri-Beri at various places far distant from each other and with no apparent connection. As in most of these places rice is imported, it is of interest to determine if these variations correspond with the rice in use. A comparison of the returns shows that such coincidences are exceptional, even in institutions and amongst bodies of men all using Rangoon rice.

Of the bodies of men fed on Rangoon rice it will be observed that there were no cases in Selangor Gaol, average strength 500, from February to September, 1903, and no cases probably originated in the gaol till December.

In Taiping Gaol, average strength over 500, there were no cases from May, 1903, to February, 1904, and no cases probably originated in the gaol till March, 1904.

During the same year there were no less than 129 cases amongst the 500 Sinkehs at Sungei Lembing.

At the Singapore Gaol, average strength nearly 1000, Rangoon rice was used only from January 1st, so that the effect, if any, of this rice might not be evident for some months. During 1903 there were, in the first six months of the year, 41 cases, but from June to September, although several cases were admitted with the disease or developed it within two months of admission, there were no cases that probably originated in the gaol till October, 1903, but in November and December there were 53 and 55 cases, and a severe outbreak, 22.1 average per month all through 1904, instead of the low rate, average about one to two per month respectively at Taiping and Kwala Lumpur.

There is, on the whole, therefore, no such evidence of variation in the occurrence of Beri-Beri as would support a theory of a common origin in the rice crop of a certain year in a certain country.

The general public use both Rangoon and Siamese rice, and the same is true of the miners, certain isolated bodies of men, such as those at Christmas Island, the Madras troops in Singapore, &c., amongst such, the occurrence was irregular. No cases occurred amongst the Madras troops in 1904. At Christmas Island there was a cessation early in 1903, followed by a severe outbreak, whilst amongst the general public in Selangor, the number of cases was less, and in Perak above the average. In the mines, the amount of the disease varied more in accordance with surroundings than in relation to anything else.

The older observations show that with the continued use of one class of rice Beri-Beri may disappear, and be absent for months or even years, and at other times be prevalent. The experience at Taiping Gaol and Kwala Lumpur, in both of which Rangoon rice has been uniformly used, are cases in point. A reference to the tables of the occurrence of Beri-Beri at these institutions is sufficient to prove this.

The theory that it is only in certain crops of rice that the disease exists in the husks, and that subsequently, during or after milling, affects the grain so that it progressively becomes more poisonous, is not disproved by these figures. It is quite possible, that whilst at one time rice from an infected crop was used, at another time the rice was not so infected.

As the rices obtained at the different prisons, at the various mines, and other institutions are obtained from various contractors, who in turn buy rice often from small importers, who again buy from various agents who deal with the growers, it is a matter of great difficulty—practically an impossibility—to trace the rice used in an institution to any one or more growers. The rice is cropped in Burmah in October and November, but it is not good for use for some months, as it is too fresh. The rice is at its best about March to June, and towards the end of the year is at its worst, as it is brittle, broken, and discoloured. Some seasonal variation should occur in institutions where inmates are fed on such rice. Unfortunately, it is no uncommon practice to dispose of the old rice by mixing it with the new, so that such seasonal variation, if it occurred, would be obscured, and the absence of this variation is no disproof of the theory. The corresponding variations in the incidence of Beri-Beri at Taiping and Kwala Lumpur Gaols and at Sungei Lembing, all using Rangoon rice, are to be compared.

The great mass of the poorer Chinese also use Rangoon rice, and though, as has been seen at Batu Gajah Gaol, the rice supplied is Siamese No. 2, still, so many of the inmates acquired the disease before admission, that any correspondencies in occurrence of Beri-Beri may also be due to this rice.

No large institutions have, as far as I am aware, used Siamese rice continuously for any lengthened period; but the Malay Police, most of the Malay peons, &c., use this rice, and in some of the mines, such as those at Rawang, Siamese rice No. 2 is supplied, and almost exclusively used by the free labour. Cases of Beri-Beri occur amongst all these classes, but not in sufficient numbers to constitute an epidemic, and no sufficiently accurate data are available for comparison as to relationship between age of rice and onset, course, or decline of an epidemic.

Rice has to be frequently imported into the Federated Malay States, as even when stored in airy and dry buildings, it rapidly deteriorates. Importers do not care to keep rice for more than six weeks in stock on account of this deterioration.

In consequence of this, rice is constantly being imported, and no large stocks are kept. The rice therefore in actual use at a large institution, even when always supplied by the same contractor, varies from month to month. All that can be stated with certainty, is that Rangoon rice supplied towards the end of the year must be old, and that supplied in the first two months of the year is either still older, or too fresh. That during the rest of the year the rice is at its best, but may be mixed with varying proportions of older rice.

It will therefore be obvious that diseased rice may be supplied at any time of the year, but that it is most likely to be supplied towards the end of the year and early in the following one.

An examination of rice nearly always shows the presence of various fungi, and other micro-organisms. In addition to vegetable parasites, nearly all rice is found to contain weevils, sometimes another beetle, and lepidopterous larvæ of five species, Tineids, or of allied genera.

The lepidoptera differ greatly in different specimens of rice.

Siamese rice is, on the whole, freer from fungi and animal parasites than any other form of rice, but neither Nos. 1, 2, or 3 are free.

The weevils are found in all grains, and seem to depend more on the storeroom than on the kind of rice.

The presence of these insects in the stored rice is not unimportant; they destroy many grains, and their excreta, webs, &c., are mixed with the rice. It is on these, or certain of the insects infesting rice, that Hose in Sarawak attempts to fix the blame.

On the assumption that Beri-Beri is due to a poison found in certain rices, it follows that this poison must be one not destroyed by boiling, and is fairly stable, possibly an organic acid. The products of the action of the ordinary moulds found in rice, on rice, either broken and raw, or made into a paste, are various organic acids such as acetic, proprionic, butyric, and occasionally oxalic, acetone commonly, alcohol rarely, and other more complex substances.

DR. BRADDON'S VIEWS.

The proofs Braddon advances are the statements that : (1) In mixed communities, and in places where Beri-Beri is prevalent, rice eaters alone are attacked; and those who eat no rice escape :—

(1.) The class of instances given are that officers of ships at sea escape, and the crews (Chinese and Malays), are attacked.

(2.) That amongst crews of several nationalities, certain races, such as Malays and Japanese, get Beri-Beri, and Europeans, Australians, and South Sea Islanders do not.

(3.) Isolated instances of various kinds.

(4.) In the epidemic in the Philippines in 1882-1883, Beri-Beri was confined to the Malays who ate rice (? fresh or recently husked). Two pauper Europeans, destitute of means to get good food, got Beri-Beri, and native patients, specially dieted on meat, escaped.

(5.) In the Philippines, the Filipino troops; in the Loochoo Islands and Korea, the Japanese troops; in Vancouver, the Japanese settlers; in Australia, the Chinese settlers and people who shared their habits; in Calcutta, the Chinese settlers; in the Soudan, the Madras sappers, &c. In each case it will be observed that the disease is limited to one race, and in each to a race and class who suffer from the disease in their own country. It is to be hoped that, in the fuller work which Dr. Braddon is producing, full and original references will be given, as in these, as in so many cases, the close association of persons of the same race are likely to lead, in the case of a moderately or slightly infectious disease, to the limitation of the disease to the one class or race.

In the General Hospital, Calcutta, in 1898, there were many cases of Beri-Beri in Indians as well as Chinese, and the class of Chinese who suffered most were the shoemakers: not, as far as I know, specially addicted to the consumption of rice.

Braddon refers to India in the terms that Beri-Beri has, generally speaking, been confined to Burmah and Madras, but omits to mention that nearly all the cases occurred in two or three regiments, that a large proportion were stationed at Singapore, and that the incidence of the disease in European troops was nearly as great as in the native troops, and much greater than in the prison population.

He further instances the Europeans in the Singapore prison who do not get Beri-Beri whilst the other prisoners do.

These prisoners are fed on bread, but the numbers are small, they work in their own gang separate from the natives, and have much fewer opportunities of acquiring a moderately contagious disease from the other prisoners. As a control, this small gang of Europeans is inferior in every way to the control experiments of either Travers or Hamilton Wright.

Even the multitude of similar instances, which Braddon states exist, do not exclude infection, mediate or intermediate, as the cause of the spread of the disease. As this factor is not excluded, the assumption that any difference in the food is the cause of the disease remains a mere assumption.

Braddon narrows down the enquiry to certain classes of rice. This excludes the rices either freshly husked or in which the husk has been split by boiling. The great argument, and to me, the only point in connection with rice or diet as an important factor in the causation of disease, is the comparative rarity of the disease amongst Tamils, as compared to the Chinese, when both are living according to their own customs.

In prisons, on the whole, there is little difference in the incidence in the two races. The conditions of life for both races are there practically identical.

Of course, in addition to the use of different rices, there are other differences in habits between the Tamils and Chinese. Even as regards

food, not only is a different rice used by the Tamils, but they use condiments such as peppers, coriander, garlic, &c., to a very large extent. Animal food is usually salt fish, goats and fowl on festivals; rarely, if ever, even the lowest caste eat pork.

There are differences also in the cooking of the rice. The water in which the rice is boiled is strained off and thrown away, instead of retained as with the Chinese. They also boil their rice longer.

They use oil baths once or twice weekly.

Braddon states that "those who eat only cured rice, never get it (*i.e.*, Beri-Beri), nor those who eat only fresh rice," and that the rice eaten by Chinese has always and invariably been "uncured"; that of the Tamils has been equally exclusively only "cured" rice.

These statements are too absolute; many Tamils, particularly those born in the country, use Siam "uncured" rice, and though instances of a Tamil getting Beri-Beri who has never eaten a grain or an occasional meal of "uncured" rice, might be impossible to find, yet cases in which only on rare, exceptional occasions has a meal of "uncured" rice been taken occur, *e.g.*:

Saminathan, Indian Tamil, aged 60, Road Coolie. Admitted to the District Hospital, Kuala Lumpur, on July 20th, 1905. States that he has been ill one month.

Numbness and weakness of hands and legs, just able to walk. Knee-jerks absent, calves tender on pressure. General œdema, most marked in legs. Urine no albumen. Has been 25 years in the Federated Malay States, always using himself Province Wellesley rice, prepared by husking after boiling. On rare occasions may have been unable to get this rice, and has then used Rangoon when up country. For many months has had no rice except the Province Wellesley "cured" rice, except on occasional visits, once or twice a month to his Overseer, he then would have a meal of Siamese rice.

He takes one Chupah, 26½ ounces of rice daily, fresh fish four times, and dried fish three times a week.

As a whole the general statements that the incidence of Beri-Beri is very small in the Tamils, and that the rice used by the Tamils is prepared differently is a correct one. Whether these two factors are in the relation of cause and effect is another matter.

The immunity of the Malay in his own village, is contrasted by Braddon with the liability of Malays to contract the disease under other circumstances, which include living on imported rice prepared without previous boiling.

According to Dr. Braddon the degree to which particular parcels of rice would be poisonous will vary with—

(1.) Quantity and virulence of the agent originally present upon the corn (*padi*) when gathered. This, again, varies with such factors as maturity, or immaturity of both corn and fungus when reaped; place, season, rain, heat, and other factors affecting fungus growth.

(2.) Manner in which the *padi* is stored.

(3.) Age of grain from which the rice is prepared; and he suggests that, as with ergot, the assumed fungus or its toxic properties may increase at first but ultimately perish.

(4.) Mode of dressing or preparing rice from the padi.

If the disease is limited to the outer covering it will be liable to be eliminated by such processes as hand sorting, &c.

Special processes, as heating sufficiently to destroy fungus, may prevent any subsequent spread or increase in the padi or separated rice.

(5.) Mode of storing rice after cleaning and separating the envelopes.

(6.) Length of exposure of rice after cleaning. The effects of this exposure varying with conditions, such as temperature, light, moisture, &c.

He concludes that—

The incidence of Beri-Beri amongst rice eaters will therefore vary.

(1.) It will be most frequent amongst those whose rice is subjected to no process that could "cure" the rice, *i.e.*, destroy the fungus or its spores in the outer coat of the padi.

(2.) It will be less frequent amongst those who eat grain freshly husked and carefully sorted and cleansed.

(3.) It will occur least amongst those who eat rice sterilized before husking.

Further—

(1.) The incidence of the disease will vary directly with the quantity eaten either absolutely, or relatively, to other food or other circumstances.

(2.) The course of the disease will be aggravated by continued use of the same (noxious) rice, and cease on its discontinuance.

(3.) There will be no fixed "latent period." The term of exposure to, or absorption of, the poison before the effects are produced, will vary with the individual and the dose, and therefore be irregular.

(4.) It will not result from mere association with those afflicted (contagion or infection), unless the food also is shared.

(5.) Epidemics of Beri-Beri both begin and end abruptly, without any change in outward circumstances to explain the event—apart from change in the food supply.

(6.) That rice, as an annual crop, is conditioned by season, and that, therefore, there may be apparent seasonal and annual variation in the prevalence of Beri-Beri, depending on the amount of the disease (assumed to be a fungus), in the successive crops, and the time of year at which they come into consumption.

Dr. Braddon considers the question of Latent Period at some length, in his abstract, and contrasts Beri-Beri with the admittedly infectious diseases, by which I presume he means such as influenza, typhoid, measles, &c.; but not leprosy and tuberculosis. In such, acute illness is produced in a short period, usually about 14 days after exposure to infection. In "intoxications," there is no definite incubation period, as there is nothing to incubate, but the length of time, before the development of symptoms, depends

to some extent on the resistance of the individual, but much more on the dose taken and the frequency of its administrations.

He claims Beri-Beri as an intoxication or poisoning, the result of unequal doses irregularly consumed, and decides that there can be no fixed or even approximately fixed period before the development of the symptoms. He considers, however, that the instances given as the result of a short exposure are fallacious, as in such cases, though the actual result may be precipitated by the last dose or few doses or other conditions, the real cause is the accumulative effect of a preceding course of the poison, and compares this with the onset of delirium tremens in a chronic alcoholic as the result of shock, an extra drinking bout, some other disease, &c.

He objects therefore to considering that on board ship the latent period is no longer than the term elapsing from the commencement of the particular voyage to the date of the attack of Beri-Beri.

He states that it is a singular fact that it is in regard to ship epidemics only that much evidence is offered in support of the contention that Beri-Beri is usually rapidly produced, and that "everywhere on land," especially with regard to the inmates of gaols, the evidence is overwhelming that a very long period is necessary before the disease actually appears. Both these statements are erroneous, and particularly so that referring to the incidence of the disease in the prisons of the Malay peninsula, *vide* Table 5 and Reports by Travers, Durham, Wright, &c., quoted elsewhere.

Braddon concludes that he has proved, "among rice eaters, Beri-Beri depends on the sort of rice eaten. Stale 'uncured' rice produces it. Fresh and 'cured' rices do not."

This important point is well deserving of proof, or almost equally important, of disproof. Many of the facts adduced by Braddon could be explained equally well on the assumption of a low degree of mediate or intermediate infectivity, and others of them on the assumption that in the better fed the susceptibility is less.

The observations in the gaols in Kwala Lumpur, it is contended by Dr. Braddon, are inconclusive, and it is admitted that further information as to the periods during which each prisoner was under observation in each class, is necessary to estimate how far the control experiment was reliable. Without this the proportion of those used as controls who can be considered as effective controls, is disputable.

That a similar fallacy exists in many of the observations quoted by Braddon is no argument as to the absolute soundness of these observations of Travers and Hamilton Wright.

His views in brief are—

Beri-Beri varies—*Directly* with (1) amount of poisonous rice consumed, (2) amount of poison in the rice, and (3) length of time for which it is taken; and *Inversely* with (1) proportion of other food, (2) possibly with the amount of work done, and (3) differences in susceptibility to the poison, whether racial or individual.

Other conditions are not essential.

The Government of the Federated Malay States offered Dr. Braddon the use of any of the larger prisons for a critical experiment, in order to prove his case.

Every facility was given, the selection of the rices placed in his hands, and such modifications in dietary and other conditions as he thought necessary, were allowed. A considerable sum of money was allotted for the preliminary expenses, and Dr. Braddon was to be in charge of the experiment and to act as the responsible Medical Officer of the gaol he selected.

There had been little Beri-Beri originating in the gaol which he considered most suitable since 1902, and therefore not only were a large proportion of the prisoners persons who had not previously had Beri-Beri, nor were liable to relapses, but the chances of infection, the rival theory, were small.

Dr. Braddon, however, considered that the experiment was not likely to be attended with success except at a time when the number of cases was considerable, and therefore declined to attempt the experiment at the time, as he did not consider the conditions suitable.

No observations, in my opinion, can be admitted as conclusive unless the question of infection by imported cases, possibly only by those in the early stages of the disease, can be excluded.

Dr. Braddon attempts to prove that "among eaters of uncured rice, the case incidence and mortality of Beri-Beri vary directly with the quantity (absolute or relative) of rice eaten."

He qualifies this statement by pointing out that it is the amount of poison in the rice that is of importance; but as this has not been identified, the direct measurement is impossible.

He considers that in a sufficiently large series the degree of concentration of this assumed poison in the different samples may be disregarded, and the rice itself considered as the direct intoxicating agent, and that then the effect as to Beri-Beri will depend, other things being equal, upon the quantity of rice eaten.

Though no direct experiment has been made to prove this, he considers that the variation of Beri-Beri with the quantity of rice eaten has been demonstrated amongst the Chinese population of the Federated Malay States.

He states that in all except business relations, and authority, individuals of all positions associate as equals. There is no condition, habit nor custom, which may be supposed to be able to affect sanitation or hygiene which is not absolutely the same for all, with again the one exception. That exceptional condition is *wealth* and such *differences in diet* as wealth commands.

Whilst admitting, with Dr. Braddon, that social equality is a striking feature in Chinese life, I do not at all consider that there are no factors other than the differences in the amount of rice eaten between the wealthier and the poorer Chinese; there are serious differences which affect the exposure to infection and immunity or susceptibility to disease.

The divisions into classes made by Dr. Braddon from the dietetic point of view, are :—

Labourers.—(1) Sinkheh, *i.e.*, new comers, coolies under indenture ; (2) Laukheh, *i.e.*, old arrival coolies who re-engage to work after having served their first indenture, at an increased wage ; (3) free coolies who work independently as miners, planters, ricksha pullers, servants, &c.

Master workmen, headmen and merchants.

Of these classes the first and second receive rations, but can supplement them in the first case to a small extent, and in the second to a greater extent. The diet of the free coolie varies, he may be underfed if poor, but selects his own diet as far as his means go. The master workmen, &c., are comparatively well-to-do and feed well, eat a liberal and varied diet, and rice forms therefore a smaller proportion of it.

The Sinkhehs no doubt suffer most severely from Beri-Beri ; the Laukhehs and free coolies much less so, and the wealthier Chinese are rarely attacked.

One fallacy connected with this argument is well shown at Sungei Lembing. The incidence of the disease is heaviest amongst the Sinkhehs during the *first six months* of their indenture, during the next six months, though still Sinkhehs and fed as before, the incidence is much less. As a rule it is little more than amongst the Laukhehs, *i.e.*, continued residence without any change in the diet diminishes the liability to the disease in places and among people where cases of the disease are numerous. Whether immunity has been acquired by slight or abortive attacks, or whether the residue are comparatively insusceptible, naturally, to the disease, may be doubted.

Braddon quotes the experience in the gaols under British control as instances of a community apparently well cared for, and states that the prisoners are placed under conditions infinitely more favourable to health and better protected against diseases spread by infection, than when free. In some respects, no doubt, this is correct : but the fact that the prisoners awaiting trial, and in many cases, for the first night after sentence, some 24 men may be, and frequently are, locked in a common cell, where they all feed, sleep, defecate and micturate, is a condition more favourable to the spread of disease by infection than any condition outside the gaol, and the fact that some of the occupants are changed each night, provides for the constant introduction of disease.

In Singapore, where the prisoners spend the first two nights in such association cells, Beri-Beri is rarely absent : in Taiping, where there is more subdivision of the fresh prisoners, Beri-Beri is more often absent than in any other large gaol in the Federated Malay States.

Other sanitary defects exist.

No connection can be traced, with any degree of exactitude, between the consumption of rice at the Kwala Lumper or Taiping gaols and the amount of Beri-Beri.

The amount of rice supplied to labourers is usually not limited, though the amounts of other articles of diet are. The average consumption in an ordinary liberal but cheap diet appears to be about 24 ozs. per diem.

Some individuals eat more, and if abundant other food, either vegetable or animal, is provided, less rice is eaten.

PRISON DIETS.

In prison diets there is no chance of altering or substituting the diet, what is given has to be taken or left.

The diets in the Kwala Lumpur prison and in the one at Singapore are here given. The diet at Taiping has been changed less than that at Kwala Lumpur, and differs little from that now in use at Kwala Lumpur. On the present diet there has been both in 1902 the largest amount of Beri-Beri, and 1903 to 1905 the smallest of recent years. The same diets were used during the first epidemic of 1895 and the first interval, with a low rate, that of 1898.

The diets in the lower columns are those which all short-sentence prisoners, and the long-sentence prisoners, during the first six months of their imprisonment, received up to 1904. They now remain on these diets for the first twelve months of their imprisonment.

In these tables I have not included punishment diet, bread or rice and water, nor certain special diets which affect a comparatively small proportion of the prisoners.

| | Prison Diets (Kwala Lumpur Gaol) per week. | | | | | Singapore Gaol,
1903-1905. | |
|--------------------|--|-------|-------|-------|------------|-------------------------------|----------|
| | 1892. | 1893. | 1895. | 1899. | 1900-1905. | | |
| ORDINARY. | | | | | | | |
| Rice | 149½ | 98 | 133 | 133 | 147 | 126 | |
| Salt Fish | 12 | 24 | 28 | 28 | 24 | 18 | |
| Fresh Meat | 21½ | 6 | 42 | 42 | 18 | 32 | |
| Vegetables | 36½ | 49 | 49 | 49 | 49 | 21 | |
| Beans | 46½ | 35 | 14 | 14 | 14 | 8 | |
| Bread | 37½ | 31½ | 31½ | 31½ | ... | 12 | |
| TOTAL ... | 303 | 243½ | 297½ | 297½ | 252 | 217 | |
| Percentage of Rice | 49% | 40% | 44% | 44% | 58% | 58% | |
| PENAL. | | | | | | | |
| Rice | 149½ | 140 | 140 | 140 | 154 | B
140 | C
140 |
| Bread | 37½ | 30½ | 30½ | 30½ | ... | 28 | 28 |
| Beans | ... | 35 | 35 | 35 | 14 | 14 | 35 |
| Vegetables | ... | 28 | 28 | 28 | 35 | 28 | 28 |
| Salt Fish | ... | ... | ... | ... | ... | 28 | ... |
| TOTAL ... | 186½ | 233½ | 233½ | 233½ | 203 | 238 | 231 |
| Percentage of Rice | 80% | 59% | 59% | 59% | 75% | 58% | 60% |

Instances are given by Dr. Braddon of the disappearance of Beri-Beri when the amount of rice is reduced or the quality changed ; and to these can be added a great diminution in the amount of Beri-Beri in the Singapore Gaol, at the end of 1904, when "Bengal" rice (grown in Siam and cured at Penang) was substituted for Rangoon rice ; but similar, or even more marked, was the diminution of Beri-Beri in Kwala Lumpur in 1903, without any change in the diet, when the introduction from without of fresh cases ceased.

The results of withdrawing rice from the diet of patients with Beri-Beri is not altogether to the point. The best results, as regards treatment, in a disease where there is a great tendency to dilation of the stomach and liability to cardiac failure can not be expected when a bulky food, such as rice, is given.

The substitution of other rices and other grains have given different results in the hands of different observers, as might be expected where the mortality depends so much on other complicating diseases.

The substitution of barley and bread for rice in one of the two wards set aside for Beri-Beri, in Kwala Lumpur, was as follows :—

| | | Cured or Relieved. | Not Relieved. | Died. |
|-----------------|----|--------------------|---------------|-------|
| On barley | 37 | 25 | 7 | 5 |
| On Rangoon rice | 68 | 53 | 6 | 9 |

Some of the deaths of those fed on barley, 13·4 per cent., and of those fed on rice, 13·2 per cent., occurred from complications. The experiment is still in progress.

Instances are given in which it is stated that no possible theory of infection could account for the incidence of the disease, *e.g.*, bachelors alone, members only of one religion, or of one race. These are as readily explicable on the ground of infection of a certain type, as on that of a certain diet.

Instances are given by Braddon of the tendency of the disease to fluctuate in the same way at places far apart, in regions as far apart as Europe, America, and the Congo territory. This he explains by the simultaneous consumption of a certain quality of rice—a certain crop—distributed all over the world from Burmah and Siam, the great exporting rice countries.

Instances of such coincidences certainly occur, but a comparison of, say, Kwala Lumpur, Singapore, Batu Gajah, Taiping Gaols, and of Sungei Lembing shows that there is no such universal correspondence as Braddon claims. The gaol epidemic at Mombasa Prison was in 1900 only, though supplied with Rangoon rice before and after. The Suva Gaol, Fiji, also supplied with "uncured" rice, has been absolutely free from Beri-Beri in all these periods.

Braddon gives instances of Beri-Beri in animals resulting from the consumption of rice. These have been considered elsewhere, but one of the instances given, the equine paraplegia, is of a different class altogether, as the lesions are definitely a spinal sclerosis.

Braddon urges that as regards some of the epidemics outside the tropics, it may be doubted whether they are true Beri-Beri, but considers it more probable that they are true Beri-Beri, and result either from the consumption (a) of rice or the products of rice (glucose) consumed indirectly, in quite unsuspected guise, as an adulterant or substitute for other grains, or (b) from

the presence, in other cereals, of the specific poison engendered by the same assumed agent which (according to Dr. Braddon, certainly,) infests and poisons rice.

Whether rice in such unsuspected guises is likely to exceed 1 lb. per diem, the amount which Braddon considers will cause Beri-Beri, may be doubted.

It will, I think, be seen that the proofs given by Braddon are not as absolute as they are claimed to be. The "Fungus" in the rice is assumed to exist; a "poison" is assumed to be formed, and various assumptions are made to explain any difficulties that arise in the application of his hypothesis to the explanation of facts. Few of the proofs given definitely exclude infection, and the facts and figures quoted for Malaya, are at variance with those of other observers.

Haviland, who also is of opinion that rice is the essential factor in the causation of Beri-Beri, defines the classes of rice used in the Malay Peninsula as the so-called *Local rice* eaten by the Malays and Tamils, and the *imported rices* from Rangoon and Siam, eaten by the Chinese. Beri-Beri, as he states, is rare among the Malays and Tamils, and common amongst the Chinese.

He discusses the question of the preparation of the Tamil rice by steaming "*à la Bengal*," as carried out in Penang, "curing" as Braddon calls it, and points out the partial nature of the process, and that after the process there is every essential for the growth of Fungi and Moulds and for the formation of toxins—Moisture, Heat, and Darkness. He attributes much of the sickness amongst the Tamils to the consumption of rice so prepared.

It is certain that the process, which consists in soaking the padi in water for 24 hours or so, and passing steam, not under pressure, into the closed receptacle in which the rice is placed for ten minutes or less, falls very far short of the treatment required for sterilization, or even the destruction of spores present in the husk. Such padi, received in a sterilized test-tube, develops an abundant crop of fungi. Rice, so prepared, yields a larger variety of moulds and bacteria than rice prepared, in the local fashion, by the Malays, or than the imported rices from Siam or Rangoon.

From such considerations, Haviland was led to attach little importance to the mode of preparation of the rice and more to the place where the rice was grown, and to suppose that the local rices, grown as they are in districts where Beri-Beri is not endemic, however treated, will not produce Beri-Beri in the consumers. The rice imported is, in fact, grown in districts in Rangoon and Siam where Beri-Beri is endemic, and he considers that the rice so grown produces Beri-Beri.

He further suggests that such rice becomes more dangerous and more liable to produce Beri-Beri if badly kept, or kept too long.

The differentiation of the rices, like that of Braddon, affords an explanation which, *primâ facie*, explains the difference in the incidence of Beri-Beri amongst Malays, Tamils, and Chinese. Like Braddon's theory, on closer enquiry, it appears less satisfactory. Much of the so-called Siamese rice is also prepared in Penang from local rice, and this rice is that supplied to Batu

Gajah Gaol, where a certain number of persons acquire the disease in prison, though most of the cases that occur may have been admitted with the disease in its earlier stages.

The great differences in the incidence of the disease in prisons, all supplied with Rangoon rice, are not satisfactorily explained under either of these theories.

It is a matter of great difficulty to trace the ultimate destination of the rice imported from Rangoon. As only a small portion of the rice is supposed to be infected, it would not follow that this should be evenly divided, or that people receiving this rice and taking equal amounts of it in different countries should equally suffer. A certain degree of correspondence might be expected, but very little trace of this can be observed.

The rice is exported in large quantities to India, Singapore, and China, but much of this is not for consumption in these countries, but is again exported. From Bombay, Rangoon rice is exported all down the East Coast of Africa, and from Calcutta by the coolie ships to the West Indies.

From Singapore the rice is distributed all over the Malay States, and to many of the islands in the Malay Peninsula, and this consignment is the one most open to suspicion.

Smaller quantities of rice are sent direct to Africa, America, and England, but how much of this is for further export, cannot be ascertained.

Braddon instances epidemics occurring simultaneously in many parts of the world. It is not difficult to find correspondencies or divergencies.

Taking the Federated Malay States and Straits Settlements, divergencies exceed correspondencies, as is seen by reference to tables.

Durham instances the almost simultaneous "disappearance" of Beri-Beri in the Gaol and Lunatic Asylum, Singapore, in the Pudooh Gaol, and on Christmas Island, in 1903, and suggests the doubt as to whether this could be more than a mere coincidence. The necessity for the introduction of fresh cases in gaol for the maintenance of an epidemic suggests the probable explanation, and the comparative absence of Beri-Beri among Singapore vagrants, as the common cause.

Singapore and Penang, it must be remembered, are great distributing centres of disease. They are probably amongst the least sanitary towns in the East under British control, as is shown by the high mortality. As adult males in the healthiest period of life preponderate, and indigenous Malaria is rare.

Little control is exercised over the exportation of disease. Even where there is nominal control, as in the exportation of cattle, the measures taken are often insufficient, *vide* Rinderpest (Studies Inst. Med. Research, No. 3, Pt. 4).

No Medical Officer of Health is resident in any of the ports of the Malay States; and at Sungei Lembing, occasional cases occur so soon after arrival, that infection must have taken place in Singapore.

It is not, therefore, a matter of surprise that with a diminution of the amount of this disease in Singapore, a place like Christmas Island should share this freedom with the Public Institutions in Singapore.

In Fiji, where "uncured" rice is used, there have been no cases at all, except amongst the Chinese, since the Japanese left the country.

At Mombasa, where Rangoon "uncured" rice was used, there was an outbreak in the gaol in 1900 only. Outbreaks have occurred amongst certain bodies of men from time to time in East Africa.

In other parts of the world to which the "uncured" rice was exported, there has been either no Beri-Beri, or in places where Beri-Beri frequently occurs, it has varied in amount from year to year, but the variations in one place do not correspond with those in others.

The testimony of merchants and others in the Federated Malay States is definite, that rice does not keep well. In Burmah also, rice keeps badly, and is exported as soon as possible after it is milled.

In these countries, only one crop of rice is grown in each year, and is harvested in the last three months in the year, usually October or November. The fresh rice is not popular, and it is at its best when about three months old; after that, it steadily deteriorates, so that the rice in the last quarter of the year and in the first quarter of the following year, is at its worst. The cheapest rice, that supplied to the poorest classes, is either in whole or in part, that kept longest.

There is a widespread belief shared by the higher European officials, that Beri-Beri is worse during this part of the year, *i.e.*, the last and first quarters, the period of the S.W. Monsoon.

In Hamilton Wright's experiment, the majority of the cases occurred in part of that period. From October to February, both inclusive, there were 73 out of the 90 cases in Party No. 2, and 144 out of 183 cases occurred in the same five months. The experiment lasted 11 months.

This carefully conducted experiment, if it stood alone, might be considered conclusive, but a reference to the tables for Kwala Lumpur Prison shows that in the following six months the cases were most numerous; that the maximum was reached in September; and that in November, December, and January, the cases—35, 55, and 22—were less numerous than the smallest number recorded in the previous six months—67 in July; and that, if first attacks only are considered, during November, December, and January, there were only 21; in October, 32; and in the five months previous, 205; or an average of 41 per month.

Taking all the prisons into consideration, it will be seen that the maximum number of cases may occur in any month, but that there is a tendency, when there is an epidemic, for this to be most severe in the winter months. An epidemic has a slight tendency to increase during the season when the rice is at its worst, and the weather colder and damper.

On the East Coast the seasonal differences are much more marked, and the differences in the occurrence of Beri-Beri are also greater; but so are the differences in the rice as communications are difficult, and consequently it has to be stored in larger bulk, and kept longer than in the dry season.

The seasonal variations are, however, no greater than in certain infective diseases, and I do not think they are more than could be accounted for by the aggravation of the symptoms in cold or wet weather causing the milder

cases which occur both in prisons, as pointed out by Durham and others, and in the mines, as I observed at Sungei Lembing, to apply for medical aid.

The seasonal variations are certainly not such as would be expected to occur if the disease were caused by rice which had deteriorated in keeping, as such rice from 3 to 6 months old, and from 9 to 12 or even 15 months, would show most material differences, instead of the slight or doubtful differences observed on the West Coast and in Singapore.

Isolated examples that seem inexplicable on the hypothesis of a poison in the rice, are given by many observers. Travers' observations on the incidence of the disease in the old and new prisons in Kwala Lumpur in 1896, and Wright's Party, No. 3, in his experiment in 1901-2, are instances in point, and are only prevented from being decisive by the small number of controls, average about 40 in each, and the inclusion of an unknown proportion who may have been under the conditions of the experiment for too short a period.

Durham gives several instructive examples from his experiences in Christmas Island. He points out that all the rice eaters obtained rice of the same quality from the same store, yet it was the coolie only who suffered. At one time there was a severe outbreak among a party of Tamils, but this was not coincident with any recrudescence amongst the Chinese coolies who lived about a mile away. These Tamils ate in company with the Malays, and though other food was different, the rice was actually taken out of the same pot of cooked rice, and the Malays did not suffer.

Even in districts where Beri-Beri is rife, large parties consuming rice, imported for general use, escape, though the rice is that ordinarily used in that district. Of such, the Tronoh Mines are an example :—

| Tronoh Mines.
1904. | | Indian Coolies. | | Chinese. |
|-------------------------|--------|-----------------|-----|-----------|
| Average number employed | ... | 300 | ... | 1,350 |
| Beri-Beri | | nil | ... | nil |
| Rice | | "Bengal" | ... | "Rangoon" |

Dr. Fox informs me that during the past two years there have been no cases of Beri-Beri amongst the men employed in these mines. Most of the work is underground, and the men are nearly all working in water. No Sinkhs are employed, the men are more or less picked men and are well paid.

In my opinion the *prima facie* case, strong though it appears, breaks down completely on close investigation. The possibility of infection, direct or intermediate, has not been fully considered.

The natural progress of an epidemic with the sharp period of decline, when the more susceptible persons have been attacked, will explain some of the instances of a beneficial effect from change of rice. The change is made when the worst of the outbreak is passed. In others, alterations in dieting have been associated with other hygienic improvements, and in others, with a cessation of introduction of chances of infection.

An inferior diet and bad hygienic conditions may, in groups of susceptible persons, promote the diffusion of Beri-Beri when once the disease is

introduced ; but the hypothesis that diet or any article of diet, sound or unsound, can originate the disease has in my opinion no facts to support it.

That an improvement in the conditions of life is an important element in causing a diminution in the amount and severity of Beri-Beri, the general testimony of unbiased observers confirms, and amongst these improvements a varied and nutritious diet takes a high place.

The cooking of the food in the Kwala Lumpur Gaol is thorough. It is sterile after cooking, and this shows that the disease is not introduced with the food, nor at the time of distribution is it contained in it, if the agent be either a living organism or a poison readily decomposed. Further, cooks in prison, as a rule, escape entirely, or to a larger extent than other classes of prisoners.

Hamilton Wright shows clearly that the cooked food is above suspicion of containing any living organism when distributed to the prisoners.

He holds, however, that subsequent to this distribution, there are chances of infecting the food by the prisoners, as their fingers may be contaminated by the dust, &c., from the cells. Other lesions described include certain mouth and pharyngeal conditions in which also an infective agent might obtain access by the mouth and possibly by food infected after distribution.

As these views are based on the theories of the causation of the disease rather than on direct observation, it is well to consider them in some detail. These conditions of the alimentary tract are considered as primary lesions, and the neuritis as a secondary result induced by absorption of toxins formed at the site of these lesions.

Sungei Lembing was the only mining centre sufficiently isolated for satisfactory enquiries. The outbreaks there were amongst susceptible gangs of men, introduced into a place where a constant supply of cases were maintained. The members of each gang acquired the disease, and so maintained a constant supply of infective persons.

A gastro-duodenitis has been described as the primary lesion by Dr. Hamilton Wright, and the same *rôle* is suggested by Dr. H. E. Durham to the pharyngeal condition.

Dr. Hamilton Wright goes so far as to propose the limitation of the term Beri-Beri to this intestinal condition, and to describe what is at present usually known as Beri-Beri as post Beri-Beric neuritis. He shows from detailed descriptions of the microscopic changes in the nerves and cerebro-spinal system, that essentially the nerve lesions are general and similar in character to the lesions produced by toxins. The toxine, he believes, is formed at the site of the gastro-duodenitis. He describes this condition as demonstrable in all cases where death occurs early in the course of the disease.

In an analysis of his cases he also shows that a history of some digestive disturbance is to be obtained in all cases of Beri-Beri. This, in my opinion, was the weak point in the argument, as native histories are unreliable, and the natives (from whom the histories were obtained), are quick to detect the answer that is wanted, and obliging enough to answer in the way that they

think will be most pleasing, without any regard to truth. The native, also, who primarily obtained many of the histories, was subsequently shown to be thoroughly untrustworthy, but in all essential points Dr. Hamilton Wright claims that he confirmed these histories.

The actual symptoms described by Hamilton Wright are (1) loss of appetite and dislike of solid food, followed in a few hours by (2) a dull pain or a dull oppressive feeling referred to the epigastrium. This was intensified by deep pressure over the epigastric region. A more or less distinct bulging of the epigastrium rapidly followed this feeling. At this stage no complaints referable to the nervous system are made, but a close examination will, according to Dr. Wright, generally reveal areas of anæsthesia or hyperæsthesia. The patella reflexes may be exaggerated.

It is not difficult to find that some cases give histories that are in accordance with those given by Dr. Hamilton Wright's patients. One patient, for instance, admitted for an ulcer, was seen by me two days before admission to Hospital, when his appetite was good and knee-jerks normal. He had vomiting for two days after admission, with epigastric pain, and took less than $\frac{1}{3}$ th of the usual amount of food. He also had headache. Three days after admission, *i.e.*, next day, his knee-jerks were markedly increased, and he developed a sharp attack of Beri-Beri, and died on the 21st day of the disease. Such histories are, however, the exception, and histories of loss of appetite, epigastric fulness, and occasionally of vomiting, are not uncommon from dyspepsia, anæmia, or malaria.

In many cases the onset of Beri-Beri appears to be gradual, and the signs of the disease or of the post Beri-Beric condition to develop, not only without any definite illness, but without the patient being aware of any illness. Amongst those observed at work and merely showing anæmia of a moderate degree and without any œdema, altered cardiac rhythm, loss of appetite, or epigastric tenderness, there was one case with a loss of the patella reflex. Two weeks later this person was admitted with a mild but definite attack of Beri-Beri, the only complaint in the intervening period had been of fever; in another similar case the disease was only diagnosable nine days after my inspection, though the patella reflex was absent when I saw him. In the intervening period there was no definite illness complained of, and in both cases, at the time of inspection, it was stated that the men ate their food well, and had no complaints. These cases had been no more than two months in the country, so that it is probable that they were the first attacks of the disease. Of the Sinkehs examined, four, in whom the patella reflex was absent, and one, in whom it was exaggerated, were admitted for Beri-Beri within two months, and in none of these was there any previous history of the disease or of any gastrointestinal morbid condition at the time of my examination.

The probability is that in such cases a mild attack occurs similar to those met with in the gaol, and that it is a recrudescence or relapse for which they seek admission to Hospital: but even in these cases this mild attack is one which shows its only effects on the nervous system, and gives no evidence of a prodromal intestinal or other disease.

In connection with these prodromata of what is usually called Beri-Beri, three main questions arise. The first purely of practical value is—

- (1.) Is there any state of illness preceding Beri-Beri that can be diagnosed before any nerve changes are manifest?

- (2.) Of scientific interest. Is there any condition preceding Beri-Beri which, though not useful for diagnostic purposes, indicates a primary lesion?
- (3.) Is this condition really primary; or is it a secondary result either of early cardiac or nerve changes?

The only prodroma of diagnostic value is that, in a large institution, a falling-off in the consumption of food has been sometimes, but not invariably, a precursor of outbreaks of Beri-Beri. This appears to be slight and of little practical value. Cases admitted to hospitals with ulcers as well as with more serious diseases, are not detected as Beri-Beri cases till definite symptoms arise; and in the gaol, even when the authorities are carefully looking out for fresh cases, nothing occurs to lead even to a probable diagnosis till the disease is definitely present.

The symptoms described by Wright it will be observed are indefinite, and such as are often met with in dyspepsia. An examination was made of 170 Coolies at Sungei Lembing, with special reference to the early diagnosis by means of these symptoms.

These 170 Sinkehs were exclusive of those with Beri-Beri, and of those who had had it. Of these, 17 complained of loss of appetite, three of vomiting, and three had epigastric pain or tenderness. One of these developed Beri-Beri in the course of the month during which I was examining, but at the time of examination he had œdema of the legs. In two others the patella reflex could not be elicited, and in the course of the next three months neither had developed definite signs of the disease. None of the others were admitted to hospital for Beri-Beri within a month.

In cases admitted with Beri-Beri, loss of appetite is common, and vomiting occasionally does occur, whilst the distention of the epigastrium is quite a feature in the diagnosis. As these cases have other quite definite symptoms of the disease, and had usually been ill for several days, the symptoms were during the early stages of ordinary Beri-Beri, and not during a prodromal stage.

The fulness in the abdomen, tenderness in the epigastrium, and loss of appetite, are often persistent during the attack, and are not limited to the initial stage.

When the extent of the nerve lesions, and the special manner in which the pneumogastric is early affected is considered, it would be a matter of surprise if no digestive disturbances were present.

Further, in fatal cases the two commonest conditions are cardiac and gastric dilatation, both conditions directly leading to the train of symptoms described.

The congested condition of the stomach and duodenum is not met with in all early cases of Beri-Beri. It occurs in later cases in which cardiac dilatation is a marked feature. The condition is in some instances not unlike the "stasis catarrh," so common in cardiac cases. The evidence that it is a primary condition and a definite precursor to the paralysis of Beri-Beri does not appear to me to be at all conclusive. As far as my own experience goes, I have seen no person in whom the symptoms described were apparent who

had also normal knee-jerks. This indicates that the nerve lesions had already commenced. I except persons who did not develop Beri-Beri subsequently.

The congested condition of the throat and pharynx has been frequently observed, and Durham lays special stress on it and on certain micro-organisms present in his cases. According to other evidence, it is a variable symptom that may be present in a large proportion of cases in one epidemic, as in that at Bentong, and absent in another. At Sungei Lembing, only three of the cases showed any congestion, and that not in very early cases. Amongst those supposed to be healthy, it occurred in only four, and none of these developed Beri-Beri within one month of the observation.

A suggestion has been made that the mouth may be the source of the original lesion where the toxine is manufactured, and attention has been drawn to the frequency of Pyorrhœa and Periodontitis in cases of Beri-Beri, but the examination of the cases at Sungei Lembing negatives the suggestion, as there were none of these affections, and in the great majority of persons the teeth were sound, and there was no visible affection of the mouth.

In spite of the numerous observations made, I consider that no disease of any part of the body has yet been detected which stands in the same relation to Beri-Beri (post Beri-Beri neuritis) that diphtheria does to post diphtheritic neuritis, as the essential and real commencement of the disease. The frequent association of other diseases, and particularly of dysentery, are in favour of these diseases, in certain cases, precipitating the onset of the disease but not as acting as essential causes.

The short period of exposure, in a considerable proportion of the cases under three months, excludes any prolonged prodromal stage, even if there were no interval of incubation required for the development of the disease.

As far as careful enquiry and examination and a few post-mortem examinations in early cases are concerned, I can obtain no evidence of any constant gross lesion of any part of the body that can be considered as *the* primary lesion.

This however does not and cannot exclude fermentative changes in the intestinal contents. The frequency with which intestinal lesions of various types are associated with Beri-Beri types is, if anything, in favour of some alteration in the intestinal contents.

LATENT PERIOD.

It is of much importance in the consideration of this disease to determine what the period of exposure to the conditions required is needed to produce Beri-Beri; incubation period plus prodromal stage according to some of the theories; absorption of poison, according to other, is necessary.

Very different opinions have been expressed. Dr. Hamilton Wright considers that it is as short as 10 to 14 days, a usual period with infective intestinal bacterial diseases; Braddon, on the other hand, maintains that the usual length of time required for the consumption of the infected rice is seven months or more. Dr. Travers has shown that at the Kwala Lumpur Gaol, prisoners who have not previously had Beri-Beri, more often develop it after three months' imprisonment than after any other period, but that earlier cases occur.

At Sungei Lembing the results obtained more closely correspond to Travers than to those of the other observers. Hamilton Wright has shown that the nerve changes are the result of a toxine; and whether this toxine is formed inside the body or outside by bacteria, fungi, or protozoa, time must be allowed for the growth of these and for the formation and action of their products.

Hamilton Wright, considering the disease as primarily a Gastro-duodenitis, naturally suggested that the probable means of conveyance of the disease was by fæcal infection, particularly that of food after cooking.

As I have already shown, the clinical evidence of a primary stage of a diseased condition of the alimentary canal is weak. Lesions resembling those described by Wright are not uncommon, but they may be absent in the early stages and may occur in all stages of the disease.

They are not of an inflammatory nature such as we should expect to find in a primary lesion, but more such as occur as secondary lesions, hæmorrhages and congestions.

The evidence of infection *via* fæces is not strong. We do not find as a constant that there is more Beri-Beri where other diseases conveyed *via* fæces such as typhoid, some forms of dysentery, and intestinal parasitic diseases, anchyllostomiasis and the like, are most common, nor amongst races such as the Tamils, whose habits expose them so much to risks of fæcal contamination.

In some towns, such as Kwala Lumpur, where there is much fæcal contamination of the earth, and where *B. coli communis* is an ordinary inhabitant of the dust of public places, theatres, gambling farms, brothels, common lodging-houses, &c., as well as of some private houses, Beri-Beri is comparatively rare.

Hamilton Wright lays great stress on the fact that during the greater part of the 24 hours, *i.e.*, 4.30 p.m. to 5.30 a.m., all the dejecta of the prisoners are passed in the cells, but exaggerates unduly the amount of contamination of walls, bedding, &c., in a well-conducted prison. It is, I am informed, most exceptional for such fouling to take place, and I have seen no evidences of its occurrence in Kwala Lumpur Gaol. On the rare occasions when it has occurred, it is, I am informed, treated as a prison offence. In lock-ups it is not uncommon. The system of defecation in cells is an insanitary one, but for 525 cells it is difficult to prevent. Where a number of prisoners, as in the civil prison—admission ward—are confined in one cell, other provisions should and could be made. In the Kwala Lumpur Gaol there is evidence that only rarely diseases, probably due to fæcal contamination, originate in the gaol, and since the town water was supplied in 1895, there has been no outbreak of any such disease at all comparable with the outbreaks of Beri-Beri.

The evidences of the disease being conveyed in the excreta of persons suffering from Beri-Beri is not supported by the experience of this prison.

It is true that dysentery is a frequent complication of Beri-Beri, as we see it in the State hospitals, and particularly at Sungei Lembing; so also is malaria. Dysentery is so commonly the terminal stage of many diseases in

the tropics, that too much importance should not be attached to this association as indicating any common cause.

The evidence alike, therefore, of a disease of the alimentary canal as a primary lesion and of spread *via* faecal contamination has little to support it.

None of these theories at all touch the main facts, that over and over again the introduction of cases of Beri-Beri into an institution are followed by outbreaks of the disease, and that without continued introduction the outbreaks soon cease.

Admitting, as I therefore do, that the disease in any prison is derived from antecedent cases of the disease, directly or indirectly, it remains to consider the probable or possible manners in which the disease is communicated.

Infection by means of water can be excluded. Individuals of various races in towns or mines partake of the same water supply, and yet only the Chinese will be attacked. Travers' experiment in changing the water from the new gaol to the old in 1896 is conclusive on this point. The Chinese, who drink little unboiled water, suffer more severely than the Tamils and Malays in the same district who use unboiled water.

Infection through the air can also be excluded; prison warders are rarely attacked; dressers, attendants, and medical officers in hospitals also escape. Prisoners on the other hand suffer in large numbers. Whilst confined in cells there is no communication by air between the cells except through the corridors where the warders, who escape infection, are stationed.

In the day, work is carried on in open sheds or in cells, with little communication between each other. The disease is not limited to prisoners in any one gang or even in any one workshed; nearly all are affected, which could only be the case if the disease were carried for long distances through the air, and this all other evidence contradicts.

A primary lesion in the mouth or fauces would be more likely to be conveyed by inhalation of dust, &c., than by infection of food. In such case the discharges from the mouth and by expectoration would be the manner in which the *materies morbi* are disseminated. In the daily sweeping of the cells this material when disseminated might be readily inhaled.

For both these modes of infection, disinfection of the cells might be expected to produce markedly beneficial results.

Various efforts have been made. Those of Travers are of interest, but are complicated with other possible effects of extra-mural work, and as has been already shown, the periods in which he claims the most beneficial results are also those in which the number of patients admitted with the disease was least. Further, the perchloride solution was kept in galvanized iron buckets, and therefore would not be up to its nominal strength.

Travers, in 1897, having as he considered practically eliminated the influence of food supply, concluded that the disease was caused by a germ or toxine present in the gaol itself.

A Commission of three Government Surgeons was appointed to investigate the cause of the outbreak, and to make recommendations for the improvement of the health of the prisoners.

The recommendations were (1) disinfection of the gaol buildings, (2) extra-mural work. The disinfectant employed was 1 in 1,000 perchloride of mercury. The extra-mural work was levelling ground and filling up swamps at the District Hospital. This was continued from December, 1897, to January 14th, 1899. During this period there were only 80 cases, an average per month of about six, as against a monthly average of nearly 31 in the preceding six months.

In 1899, the prisoners were engaged in extra-mural and partly in intra-mural work and the admissions for the year, 73, were practically the same as during the period when they were nearly all doing extra-mural work.

In 1900, intra-mural work was resumed, and extra-mural, with the exception of a small gang, abandoned. The cases for 1900 and 1901 were 180 and 205, respectively, at the rate of 16 per month; and for 1902, up to the end of September, at the rate of 45.1 per month. From the 26th September, 1902, to January 22nd, 1904, the work was done in open sheds outside the prison walls, and there was a rapid decline in the number of *fresh* cases admitted to Hospital; in October there were 39; November, 9; December, 16, and January, 8, an average of 18 only; and in the next seven months there were no cases at all, and the total number of subsequent cases till the extra-mural work was stopped on January 22nd, was only five. One of these was not a first attack.

Since the resumption of intra-mural work there has been no extensive outbreak, but in the period of 18 months there have been, inclusive of relapses and persons admitted with the disease, 38 cases. Of these, some 22 perhaps in all acquired the disease in gaol, an average of 1.2 per month.

To the instances quoted by Travers, may be added the gangs engaged in extra-mural work at the old gaol in 1895-1896, and Party No. 3 in Dr. Hamilton Wright's experiment in 1900-1901. In neither of these instances did any cases develop amongst the parties engaged in extra-mural work.

I am not prepared to admit that extra-mural work was the sole or even the most important of the causes of the variations in the incidence of the disease, and still less to admit that these variations prove earth or ground infection. In the mines, at Christmas Island, in the Prison at Mombasa, the work was entirely extra-mural or outside the houses or living places.

The figures may show that extra-mural work is an important factor in preventing the spread of the disease amongst the inmates of a prison, and may further show that the cause of this spread is not, exclusively, a nocturnal agent.

The results of disinfection in the gaol at the end of Wright's experiments, February, 1902, are important in this connection. The cells were sprayed with formalin in solution. Dr. Wright assures me that not only was the greater part of the prison treated, but that special attention was paid to the cells which had been occupied by prisoners who had acquired the disease whilst in occupation of them.

During the period March, 1902, to January, 1903, eleven months, after this disinfection, there occurred no less than 803 cases, more than three times as many as in any previous equal period, it was therefore ineffective. Subsequent to this disinfection, the cells were treated with 1 in 1,000 perchloride of

mercury solution irregularly. A few cells were so treated each day, and in the course of three or four months the whole number of cells would have been so treated, and the process would then be repeated. At times no such disinfection would be carried out, and it is difficult to obtain any correct estimate of the regularity of the process. The decline in the number of cases towards the end of 1902, and the disappearance during the greater part of 1903, and the low rate for the rest of 1903, 1904, and the first half of 1905, are certainly not due to any energetic or regular disinfection of the cells.

No striking results appear to have been obtained anywhere by disinfection. At Christmas Island, according to Durham, the floors and bed boards were scrubbed out at first weekly, and subsequently every fortnight, with 1 in 4,000 perchloride solution. The houses were occupied by the coolies, and the coolies were the class attacked. The artisans, whose houses were not so treated, escaped entirely.

Hamilton Wright believed that the cells became infected as a result of defecation in the cells, and appears to have believed that a general change in the system had been made. A change was made, which affected a small proportion only, namely, prisoners in solitary confinement. A change of importance was made in that prisoners, instead of having all their meals in the cells, had the morning meal outside, and so the risk of contaminating the food with the dust of the cells was for that meal obviated.

I do not consider that, on the whole, changes sufficient to very markedly diminish the risks of contamination, whether by the food or by inhalation of dust, have been made, and the changes were not sufficient to account for the marked diminution in the occurrence of the disease in the gaol at Kwala Lumpur. The diminution in the number of prisoners admitted with the disease, or in the early stages of it, still appears to me to be the probable cause of the absence of any extensive outbreak.

The balance of evidence appears to me so strongly in favour of infection that a consideration of the manner in which it is spread, on the assumption that it is infectious, is required.

- (1.) The mere admission of a person suffering from Beri-Beri does not necessarily or even usually lead to the infection of persons either associating with him or in close contact.
- (2.) The admission of persons in the late or paralytic stages of the disease does not seem to be provocative of an outbreak. It is by the admission of persons apparently healthy, as in the last Penang outbreak, in the 1895-1896 or 1902 outbreaks at Kwala Lumpur, that the infection is introduced. These persons are in such an early stage that they pass their medical examination, no careless one, on admission to the prison, but are admitted, within a short period, to hospital with definite signs of Beri-Beri.
- (3.) Relapsed cases may, as in Taiping, be a source of infection; but this is rare. An epidemic dies out, though fresh susceptible uninfected individuals are constantly introduced where relapses are occurring, though not where persons in the early stages of the disease are also introduced.

- (4.) Though the disease does become generalised, and spread from inmate to inmate of a gaol to a considerable extent, this spread soon ceases, unless fresh infected cases are imported into a gaol.
- (5.) In persons living under freer conditions, as at Sungei Lembing, the converse is true, as the outbreak continues as long as susceptible individuals are introduced.
- (6.) That the disease in an isolated community may spread widely, and apparently from one person to another where no direct contact or association can be traced or can conceivably occur. Yet susceptible persons, more closely associated or working in the gang or occupying adjoining cells, may escape.

Such a series of provisions indicates that the mode of transmission is complex, and it would appear that not only are persons in the earlier stages of the disease, *i.e.*, before the advent of the more marked secondary nerve lesions, necessary for successful infection, but that these early cases also introduce some condition which enables the disease to spread.

A revision of our views on infection and infective agents is rendered necessary by the extension of our knowledge of the diseases carried by insects, malaria, yellow fever, relapsing fever, and other forms of spirilla disease in man, may be taken as instances, as well as the part played by insects in the propagation of many diseases of animals. This naturally leads to the consideration of the possibility of Beri-Beri being conveyed in a similar manner. Ship epidemics show that outbreaks can occur and spread, where most of the ordinary biting or bloodsucking insects are either absent or extremely rare.

Mosquitoes can, I think, be excluded. They are present, and if carriers, would further carry the disease from the cases originating in the gaol indefinitely, so that as long as fresh susceptible prisoners were introduced, the outbreak would continue. The freedom of European prisoners, Eurasians, warders and other officials from infection would be inexplicable.

Direct experiments are wanting on this subject. Comparatively few species of mosquitoes are found in the gaols, and these occur also in countries where Beri-Beri is unknown. There is no connection between the number of mosquitoes and the amount of disease in districts or areas where the disease is endemic. The mosquitoes found in the gaols are *Culex fatigans*, *Stegomyia scutellaris* (and in Singapore, *S. fasciata*), *M. rossi*, *M. barbirostris* and *sinensis*, the common town mosquitoes, but all found in Kwala Lumpur in far larger numbers than in the gaol. They are all hardy mosquitoes, capable of travelling considerable distances, and, if carriers, the immunity of certain classes in the gaol is inexplicable. It would not explain the immunity of certain classes of the community, *i.e.*, the Tamils, who suffer so little, when the Chinese, living, as at Sungei Besi, in proximity, escape.

Other diptera that can be excluded are *Tabanidae*, and other biting flies, partly on the same grounds as the mosquitoes, and still more because they rarely bite man, are more found in association with the larger animals, and are not met with in the larger prisons, such as Singapore and Kwala Lumpur.

The so-called "Sand-flies," various species of *Chironomus*, are very abundant in certain localities, such as the Ulu Klang, where the small mines

surrounded by jungle, are situated. A large proportion of the men working on such mines are attacked, and it is possible that there the disease is spread by these flies, but as they are not found in the gaols, if they are carriers at all, they are not the carriers in the institutions.

The ordinary bed bug, *Cimex lectularius*, the only species I have seen in Malaya, has been suggested to me as a possible carrier by J. M. Wright and Gerrard. Durham also had considered the possibility.

These pests are widely distributed, and occur on ships and in prison cells, though not in very large numbers. They are nocturnal in their habits, and remain more or less in one locality. Premising that they carry the disease, it would explain satisfactorily the limitation of the disease to certain localities, the spread of the disease in a gaol after the introduction of a case first through one grade and then through another, the persistence in a place—once the disease has gained a firm footing—for variable periods, and the abrupt cessation of the disease, although fresh and susceptible persons are constantly being introduced. It does not explain satisfactorily the scarcity of the disease amongst Tamils, as the Tamil houses are no freer from these pests than the Chinese. The Tamils themselves complain of being bitten by these animals. It was considered possible that the "oil baths" which the Tamils employ, might be some protection, but according to their own statements, these make no difference in their liability to be attacked.

The localisation of these bed bugs is not so absolute that it is possible for years to elapse before infected bugs from one Kongsu inhabited by Chinese could obtain access to an adjoining hut inhabited by Tamils.

The only remaining blood-sucking insects are the pediculi. Of these, the *Pediculus capitis* is the only common one in the Malay States.

It is found on the heads of individuals of most of the races, and is at least as common on the heads of the Tamils as on those of the Chinese.

It is not improbable, however, that the pediculi infesting one race, even when they leave one host, select the head of an individual of the same race as the original host, and that the pediculi from a Chinese host would only exceptionally be found on a Tamil, and *vice versa*.

It is well known that the colour of the pediculi in different races vary, and this difference is marked between the pediculi on Chinese and Tamils. These differences are said not to be sufficient to represent specific differences.

The variation in the appearance of pediculi in different races, such as Tamils and Chinese, are of colour and markings only. As variations in colour occur with the age or size of the parasite in both sexes, even in the same head of hair, it does not appear probable that these differences are more than an adaptation to surroundings.

The manner in which, in the same individual, these parasites select one special hairy region, shows that minor differences are important in influencing their selection, and it is natural to suppose that the pediculi from the hair of a Chinaman would prefer the head of another Chinaman to that of a Tamil, as the manner in which the hair is treated differs greatly.

The Tamils of the lower classes usually oil their hair well with Sesamum oil twice a week, and then rub it with limes or boiled leguminous fruit, and finally wash with water.

They comb their hair daily.

The hair is twisted up into a loose knot at the back of the head.

The hair of the Tamil is less coarse than that of the Chinaman, and not so straight.

The Chinese customs vary. Those fresh from China use no oil.

Most of them wash their hair about once a month, using water in which cinnamon bark and other odoriferous substances have been boiled; others use Chinese soap. Certain individuals, and as a class, the ricksha pullers, wash their hair more frequently, sometimes as often as two or three times a week. Many of the Chinese only have their hair combed when they have the other part of the head shaved, *i.e.*, three or four times a month. Occasionally, but not commonly, they may loose their hair at other times, and comb it.

After a variable time in the country, usually some years, but it may be some months, they often use oil for their hair. This is applied on shaving days only. The adoption of this custom is not invariable. Among the class frequenting the District Hospital, an enquiry made from the patients, none of whom were Sinkhehs, resulted as follows :—

Of 63 Chinese patients with Beri-Beri in the District Hospital, Kwala Lumpur, none were in the habit of oiling their hair. Only one washed it more frequently than twice a month, 26 and 23 washed it respectively twice a month and once a month, and 11 less frequently than every three months.

Of 50 other patients of a similar class, but who gave no clear history of Beri-Beri, the habits differed little. They also used no oil, but two of them washed their hair every week, and only four less frequently than every three months. The usual time with both classes of patients, those with Beri-Beri and those with other diseases, was once or twice a month, 76 and 80 per cent. respectively.

The Penang born Chinese almost invariably use oil (cocoanut oil) for their hair, and so do most of other Chinese born in the Straits Settlements. Chinese children of all classes usually have their hair oiled up to about 10 or 11 years of age. Chinese women, whether born in the country or not, all use oil and wax. The preparations they use differ a good deal according to their means, but always differ from the oil used by the men.

A barber may be of any nationality, and it is not usual for race to be taken much account of. A Hylam or Macau may go to a Hylam barber, or otherwise. Price is the main consideration. Other things being equal however, the tendency is to go to a man of their own nationality.

Infection by lice would provide a plausible explanation for the uncertain course of infection. The parasite can pass its whole life on the one host and only accidentally or incidentally leaves it to seek a fresh similar host. That this migration, though not essential, is fairly common, is shown by the large numbers of a fairly cleanly race such as the Chinese who are infested with these pests.

The Tamils rarely sleep with or in the same beds as the Chinese, and would be little liable to infection from the Chinese by their parasites, even if there were no differences in the hair sufficient for the parasites from the Chinese to prefer Chinese.

Persistence of infection in a place would last as long as infected parasites would live. Dissection of these parasites has not shown any bodies that appear to be parasites.

Experiments were made by allowing lice from persons with Beri-Beri to escape on the bodies of an Orang-Outang, monkeys of two species, from six to 20 lice were allowed to escape on each, on each occasion. The lice were collected from several patients with Beri-Beri, and early cases were used by preference. None of these had however been ill for less than a month, and some had been ill for as long as three months. The lice were kept for several hours before being allowed to escape on to the animals, so that they might attack their hosts as soon as possible. This is necessary, as the monkeys soon find and eat as many as they can, and though at first the attention of the monkeys can be distracted by feeding, this is only for a short time. Few can be found after 24 hours.

The Orang-Outang alone does not seem to be incommoded by these pests, but even from it they rapidly disappeared.

The results were negative as regards the production of any definite disease.

On other animals similar experiments were made, viz. :—White mice, rats, guinea pigs, and rabbits, with negative results in all cases. The lice, though they could not escape as the legs of the cages were immersed in paraffin, could not be found on the bodies of the animals after a few days.

Whilst realising that we do not know whether a parasite is the cause of the disease, nor that if a parasite it is an animal, nor that if such a parasite exists that it requires an intermediate host, analogies are in favour of such a view.

Of possible intermediate hosts, pediculi appear to be the most probable.

This hypothesis affords a plausible explanation of the main facts as regards racial and class incidence which are difficult to explain on any theory of infection *via* air, water, dust, or food. The rarity of the disease amongst Tamils, and its frequency amongst Chinese living under not dissimilar circumstances; the rarity of the disease amongst the better cared for and wealthier classes of Chinese, amongst the Straits-born Chinese, the Chinese servants, and the escape of the Europeans. The rarity of the disease in females, other than prostitutes, and the immunity of the Hylam servants, who usually oil their hair, are instances in point.

It further explains the loss of these differences in gaol with the exception of the Europeans. No prisoners are allowed the use of oil, and therefore the differences in the treatment of the hair are slight. The European prisoners are little associated with other prisoners, and in Singapore, except for the treadmill, do not work in the sheds used by the other races, and occupy a separate block of the buildings.

It might perhaps be expected that barbers would be more exposed to infection if this is the mode of transmission. Barbers do get it, but there is

no evidence that there is any special liability amongst them. Patients with Beri-Beri are less likely in the early stages of the disease to have their hair attended to, and so much mutual help in hairdressing is given by the poorer classes to each other, that the absence of any special incidence on the professional barbers has little importance.

The negative results in animals are of little value. It is not proved that the forms of peripheral or polyneuritis of the lower animals are true Beri-Beri.

Our knowledge of the development of parasites in intermediate or definitive hosts shows that it is essential that the parasites should be introduced into such hosts at a certain definite stage of the life of the parasite for such development to take place, and further, that between the introduction into one host and its transmission to the next, a sufficient interval must elapse for further development of the parasite to a suitable stage for transmission.

Such work therefore as has been done, has been done in the dark as to these important conditions, even if the animals used were susceptible.

More conclusive results may be anticipated if the occasion offers, where abundant fresh cases of Beri-Beri are available.

In the meantime, information could be obtained by the success or otherwise of greater attention to the care of the hair and the destruction of vermin in places and institutions where Beri-Beri is rampant and of fairly constant occurrence, such as Sungei Lembing mines and the Singapore prison.

Whatever may be the exact mode of infection, there are certain points in connection with the management of the prisons that give chances for spread of diseases infective in any way, particularly for the spread of disease by organisms or intermediate hosts, which are in close association with the definitive hosts.

All prisoners during the first night after conviction, are confined in association wards, till they have been examined by the Prison Medical Officers. If convicted on Saturday, they would spend two nights in these association wards. These association wards usually form part of the block known as the Civil Prison, and in it are also confined prisoners awaiting trial, prisoners undergoing simple imprisonment, as well as all prisoners in the interval between their conviction and the examination by the Medical Officer.

Details differ in the several prisons. In Kwala Lumpur, the association cells are large, and though the prisoners of various classes are nominally kept apart, in any time of pressure, any one class may be mixed with others.

In Taiping, the association wards are numerous, but smaller; and though the wards are often overcrowded, there is little possibility of any two classes being mixed. In Penang, the wards are large and airy. The classes are kept distinct, but sometimes prisoners are kept longer in them, as the accommodation in the cells is insufficient; so that to some extent, these wards are used for the overflow from the criminal side of the prison.

In Singapore, the Civil Prison is quite distinct. The convicted prisoners, after being confined till examined by the Medical Officer, are kept for two days in other association wards before they are drafted into cells, in

order that any infectious disease, such as Small-pox, which may develop in two or three days after conviction, may be detected and general infection prevented.

In most of these prisons the prisoners sleep on the floor, but in Singapore there are large sleeping platforms, fixed at a height of about 2ft. 6in. from the cement floor. These platforms occupy the greater part of the ward. Such places are very difficult to clean underneath.

Cleaning in most of these wards, consists in daily brushing and weekly washing. In the Kwala Lumpur Gaol, the admission wards are on the first floor. The ground floor is used for guard-room, store-room, &c. Washing has to be done with great care, and a little water used, as otherwise water will drip through into store-rooms, &c.

In all the gaols, defecation and micturition is carried on in the ward, as the prisoners are locked in from about 5 p.m. to 5.30 a.m. This system, perhaps unavoidable in separate cells, is unjustifiable in these wards, sometimes badly ventilated, in which up to 20 or 30 men may be confined.

All prisoners on admission bathe, but those awaiting trial wear their own clothes, the others receive a suit of prison clothing. Anything therefore that either is a source of infection or a carrier of infection in these clothes may be thus introduced into the prison.

Bedding.—In all prisons a blanket is provided. In some, as in Kwala Lumpur, a mat is also provided. In others, a simple wooden pillow only.

The prisoners sleep on hard-wood boards fixed to the wall, except in Penang. Where there is a wooden floor no bed boards are used.

The blankets used in the admission ward are exposed to the sun, if the weather be suitable, and re-stored, to be again issued to incoming prisoners or to those confined in cells.

In the admission ward it is possible that the same blanket may be used almost every night by a different prisoner. Some of them, *i.e.*, those awaiting trial, use their own clothes—more or less dirty—and the prison blanket. This blanket is used till it is worn out.

The blankets used in the admission ward may be issued to prisoners in cells of any grade, except in Penang.

No blankets, except in some prisons those used in hospitals, are washed or disinfected.

This arrangement would, it is obvious, entail considerable possibilities of the spread of diseases introduced by prisoners awaiting trial or recently convicted, amongst those admitted on subsequent days, and more rarely amongst prisoners who have already been in gaol for so prolonged a period that they have worn out one or more blankets. Certain skin diseases, itch, and prurigo, are fairly common amongst prisoners, and may be spread in this way. Whether Beri-Beri spreads *via* expectoration, evacuations, or by intermediate hosts, such as bugs or lice, the system provides a *modus operandi* for such communication.

In the cells, the practice varies. In Kwala Lumpur it is customary for a prisoner to take with him his bedding if his cell is changed. A prisoner

who is punished by confinement in cells does not in those cells use bedding, and when returned to the ordinary cell receives another not necessarily clean or new blanket.

In Penang, the bedding is considered as part of the cell, and when a prisoner leaves or goes to another cell, he leaves the bedding behind him, and uses what he finds in the cell he is transferred to.

In Penang, in addition to the cells being washed out weekly, the bed boards (which are moveable), are scrubbed, taken out of the cell, and sunned.

The new blankets in Penang are issued to the prisoners in the criminal prison (cells). The stored blankets for the Civil Prison, which includes the part of the gaol occupied by new prisoners, those undergoing simple imprisonment, and those awaiting trial, are kept separate, and in a separate store from those issued to prisoners in cells.

The rarity with which, in spite of frequent introductions of Beri-Beri into Penang Gaol, the disease has spread, may be in part due to the difference in the system of storing and issuing the bedding.

It is possible, therefore, considering these conditions, that an influx of persons in the early stages of Beri-Beri to a prison should infect other prisoners, provided the disease be in any sense infectious. It may readily become widely generalised from the admission wards throughout the gaol. The experience of all the prisons supports the view that prisoners so admitted are associated with both large and small outbreaks among the prisoners.

The lock-ups in the various Police Stations are possibly the sources of infection in certain cases, but probably more so in the small towns and mining districts than in Kwala Lumpur itself, as prisoners may be kept longer in the country stations.

In Kwala Lumpur, prisoners are kept in the lock-up till they have been before a magistrate, this is usually one night only, but in case of arrests on Saturday, may be two nights.

In some of the lock-ups the cell is an association cell, but in others small cells, in which one or at most two prisoners are confined, are also in use.

In these places the inmates do not bathe, they wear their own clothes, and in some of them, such as the Central Police Station, the cells are usually occupied, and may be overcrowded.

The cells are well ventilated, and are washed with water each day if they have been occupied. The floors are cement. The bed platforms are usually fixed, and thorough cleansing is impracticable. No other measures are taken for the removal of vermin.

Access to a separate latrine is allowed in some of the lock-ups, in others a latrine is provided in the cells, and the prisoners micturate into a bell-shaped cement sink in the floor. As the cement is always porous, in spite of a liberal use of Jeyes' fluid, an objectionable smell is sometimes present. The prisoners, whether remanded or convicted by the magistrate, are transferred to the Civil Prison admission ward in the Kwala Lumpur Gaol.

In a country district, the prisoners after being seen by the magistrate, may, if remanded, be sent back to the lock-up, and in the smaller districts, visited twice or three times a week by the magistrate, the prisoners are frequently kept for two or three nights in any case. In such country places it may happen occasionally that a lock-up is overcrowded, more commonly it is unoccupied.

For this and other information, I am indebted to Mr. Hatchell, Chief Police Officer.

Before considering the whole question, a brief *résumé* of the clinical aspects of the disease is requisite. No new facts have been elicited, but certain points in connection with the variations in the disease, and in its early stage, are not, in my opinion, sufficiently emphasized.

In brief, Beri-Beri as we know it, is a disease affecting large tracts of the nervous system, including the pneumogastric, the nerves supplying the limbs, but excluding the cranial motor nerves and the nerves of special senses. The mental faculties are not impaired.

EARLY SYMPTOMS.

Weakness and debility of the legs, associated with a slight degree of oedema, often most marked anteriorly in the legs; tenderness of the muscles, particularly of the calves, and deviation from normal in the patella reflex. This deviation is usually a loss or diminution. In some of the cases the patella reflexes are markedly increased, but after a few days, rarely weeks, diminish and are lost. In prisoners who apply earlier for medical aid than labourers, a much larger proportion have this increase of the patella reflex when they first present themselves for examination. In the Penang outbreak they were noted as being present, or increased in 20 out of the 36 cases at the time of admission, though subsequently they were lost in all. In labourers also, if examined whilst still at work, this increase without any other definite sign of Beri-Beri, is sometimes met with days before the affection is sufficiently severe for them to voluntarily come to hospital.

I believe, with Durham, that this preliminary exaggeration is far more common than is usually supposed, but do not consider that it is invariable. If always present it must sometimes be so transient a condition that it is overlooked.

For practical purposes I consider that if the frequency of this exaggeration is overlooked and a loss of knee-jerks considered as an essential feature in the diagnosis, many persons in the early stages of the developed neuritis will be passed by a Medical Officer as free from Beri-Beri.

In these, the earliest stages, there are usually, in addition to the alteration in the knee-jerks, other evidences of alteration in the nerves, such as weakness of the lower extremities, flaccidity of the muscles of the legs, rarely cramps, and alterations in the sense of perception in certain cutaneous areas. All these symptoms may be very slight. Associated with these changes are signs of affection of the organs supplied by the pneumogastric nerve. These signs include irregularity of cardiac rhythm, undue cardiac excitability, and sometimes definite signs of cardiac dilatation. There is also dilatation of the stomach, loss of appetite, epigastric fulness and frequently tenderness.

The exact sequence of these symptoms is uncertain. They may all precede loss of knee-jerks. These symptoms may form the sum total of mild cases of the disease. A few days' rest in bed restores the patient apparently to a condition of normal health, and there is no definite paralysis supervening. The knee-jerks are absent for some weeks. In Kwala Lumpur prison, 1903, 1904, 1905, the majority of cases admitted were of this type. In some cases the patients were re-admitted with a recurrence of the symptoms, which either passed off as rapidly as at first, or continued for a longer period, and were associated with definite if partial paralysis. One of the patients, after one relapse almost as mild as the first attack, in neither case associated with more definite cardiac symptoms or paralysis, had a second relapse without any observed premonitory signs, and died in 24 hours.

Out of 19 consecutive cases admitted and discharged during the period from November, 1903, to November, 1904, and all definitely diagnosed as Beri-Beri by the State Surgeon, Travers, and myself, five were discharged in a week or less, five in less than two weeks, and only four were detained in hospital for over a month.

These cases occurred during a period when there was very little Beri-Beri in the gaol, but such cases also occur during the big outbreaks. In 1902 Durham remarked that of the prisoners not admitted to hospital, a large proportion were affected. Among a comparatively free community such as the Sinkhehs at Sungei Lembing, several cases of a very mild type occurred in persons who continued their ordinary work and did not seek admission to hospital. At Christmas Island, where a severe form of the disease formerly raged, the disease, though still prevalent, is now of a mild type; out of 319 cases treated from January to July, 1905, only five died, under 2 per cent. In 1902 the case mortality was 13 per cent.; in 1903, 10 per cent.; and in 1904, 9 per cent.

An erroneous idea of the average severity of the disease is gained by a purely hospital experience. The cases as seen in hospital are either only the severer cases or cases of a milder nature in which intercurrent diseases such as dysentery have supervened.

There is nothing to show that these mild cases—cases so mild as to be overlooked—are not important agents in the spread of the disease in such outbreaks as some of those on board ship, in isolated communities, or even in gaols.

With our experience of the importance of ambulant cases of typhoid, too much importance can hardly be attached to these common *ambulant* cases of Beri-Beri. The classic accounts of Beri-Beri mention these mild cases, but lay too little stress on the frequency of their occurrence. In these accounts the types described are those in which more extensive, more severe, and more persistent lesions occur. In these the results of the disturbed innervation are necessarily more pronounced.

On the nerves mainly implicated, the severity of the cardiac manifestations or general muscular system depends.

In some cases the paralysis, particularly of the extensor muscles of the foot, may be very marked. In the earlier stages there is always, in my experience, oedema, it may be only slight, and if as is common, a rather firm form of oedema, it may escape detection unless firm and continued pressure is made.

In other cases the oedema may be marked and so general that the appearance of the patient simulates that of a person with the dropsy of acute nephritis. This difference in degree has led to the misleading differentiation of Beri-Beri into "wet," *i.e.*, with much oedema, and "dry," in which the paralysis alone is noted. The oedema and paralysis occur together, but the oedema often disappears in whole or part, and a case of "wet" Beri-Beri will then appear to be a case of "dry" Beri-Beri. Muscular tenderness may be extreme. It is, in a well marked case, always present. It may be only marked in large muscles such as the calves, or the small muscles such as those in the thenar and hypothenar eminences may show it to a marked degree.

The knee-jerks, even in the severe forms, where chances of early examination occur, are frequently found to be exaggerated. They may remain increased for a time, but ultimately are lost. They rarely return with disappearance of paralysis, usually not till some time after the patient appears to be in perfect health. Occasionally they are long absent when otherwise recovery appears to be complete. Travers' observations on long-sentence prisoners who had had Beri-Beri in 1902, are of interest in this connection. The 47 prisoners who were examined, were engaged in ordinary prison hard labour and appeared to be in good health. The examinations were made on August 20th, 1903. One had been discharged from hospital over one year—knee-jerk still absent; one about a year—knee-jerks free.

| Period
since Discharge
from Hospital. | No of
Cases. | Knee-jerks. |
|---|-----------------|---------------------|
| 13 months | 1 | Absent |
| 12 " | 1 | Present |
| 11 " | 6 | 5 Present, 1 Absent |
| 10 " | *17 | 16 " 1 " |
| 9 " | †4 | 4 " " |
| 8 " | ‡17 | 12 " 5 " |
| 7 " | 1 | 1 " |

*Two were weak, one increased. †One weak. ‡Three weak.

So that out of a total of 47, seven months or more out of hospital, 39 had recovered their knee-jerk, though in six they were weak, and in eight only were they still absent. In one case, nine years after the attack, and without definite intermediate recurrence, these reflexes were still absent.

The most important series of symptoms—those affecting life—are those dependent on the changes in the pneumogastric nerve and its branches.

The more this nerve is implicated, the greater the risk, even if other symptoms are trivial. It is rare for no evidence of its implication to be present, and the usual manifestations are cardiac. Irregularity of rhythm, reduplication of the first sound, abnormal spacing, are common, and these are greatly increased by movement. Attacks of cardiac dilatation occur in some cases and may prove fatal, or death may occur, without any warning, from syncope. Certain intercurrent diseases, such as malaria, are very likely to prove fatal even in cases where, before the onset of the malarial attack, the heart symptoms were not severe.

Dilatation of all the cavities is the rule. In the more chronic cases this may be associated with a slight degree of hypertrophy. The average weight of such hearts, free from blood and clot in 34 cases, was 318·81 grammes.

The epigastric distress is in part probably referable to the cardiac condition, but there is little doubt that the innervation of the stomach is also affected. This is usually shown by the great tendency to dilatation. If food or fluid be allowed to be taken in excess this dilatation is extreme. Congestion and ecchymoses are common. It occurred in 13 cases out of the 34. There were none of them very early cases, and some in which it occurred were of old standing. In recent cases, not of a very severe type, terminating fatally quite early in the disease, from combination with intercurrent disease, the condition was in several instances absent. The laryngeal muscles are sometimes affected, and result in partial or complete paralysis of the vocal cords. Such cases are usually associated with marked cardiac affection, and in them the prognosis is unfavourable.

The rarest form is that in which the development of the symptoms is so rapid that the patient dies within 24 hours or even less of the onset of serious illness.

The nerve changes are, according to Hamilton Wright, such as are only met with as the result of a toxemia, and are not unlike those in post-diphtheritic paralysis. It is on account of this similarity that he proposes to call the condition as ordinarily seen post Beri-Beri neuritis, reserving for the assumed primary lesion, the term Beri-Beri. This lesion he believed to be the gastroduodenitis already mentioned.

There is no constant gross lesion nor any group of symptoms that distinctly point to a primary lesion, and we are forced to assume therefore, that the primary lesion or prodromal condition, is not associated with either a gross lesion or with any definite marked symptoms.

Hamilton Wright considers that the period of exposure necessary for the development of the nerve changes, or secondary lesions, may be as short as 12 to 14 days. The Penang outbreak indicates a period between 10 and 20 days, and the general experience is that under three months' exposure, and therefore a shorter period of incubation, is the common period, and that cases develop in two months, or even earlier in a few cases.

During this period, a regular sequence of events must take place if the disease is an infectious one or due to any form of micro-organism—animal or vegetable—producing in the body a toxine.

(1) Infection must take place, then (2) an incubation period follows ; (3) a primary lesion or stage during which the toxine is manufactured ; and (4) a period, possibly very short, during which this toxine affects the nerves sufficiently to produce symptoms.

The month or less is not an impossibly short time for this series of events. There is no impossibility in the third stage, that of the growth of the organism which develops the poison, being imperceptible, and the variations in the mode of onset sudden or gradual and of varying severity, are such as are met with in other diseases, where toxins are formed by micro-organism.

Unless the patient dies rapidly, in uncomplicated cases recovery is the rule, and the subsequent decline in the severity of the symptoms might be explicable either on the hypothesis that the primary cause soon ceases to exist, or if a more persistent organism, that anti-toxines are formed which neutralize the effect of their products.

A comparison with other diseases with known causation, in which a special portion of the nervous system is of interest. Diphtheria is the most closely allied clinically, in that the resemblances of post-diphtheritic paralysis, though affecting usually different groups of nerves, to the paralysis of Beri-Beri, are great.

The primary lesion in diphtheria, though usually the throat, may be in any part of the exposed cutaneous or mucous surfaces. The primary lesion may be very slight, and yet severe paralytic symptoms rapidly develop.

Hydrophobia need hardly be mentioned as the living cause, though unknown, it undoubtedly lives in the nerve tract mainly implicated. The period however for the whole series of events in Beri-Beri, even taking the shortest estimate of the time between first exposure and development of the disease, is not shorter than the similar period required for the development of rabies in many animals.

Tetanus is more to the point, though the portion of the cerebro-spinal system on which the toxine acts is so different. Here the "primary lesion" may be insignificant, completely overlooked by the patient, and either very difficult to detect or even impossible to find on most careful investigation. In this disease the toxine is elaborated in a part well removed from the nerve centres mainly attacked, and it is only when the poison has sufficiently accumulated, which requires a variable period, often shorter than any period required for Beri-Beri, that the symptoms manifest themselves.

In this disease, whether the primary cause is removed or not, even if the wounded part in which the organisms are growing is amputated, the effect of the poison continues, and the patient either dies rapidly or recovers slowly.

The rarer forms of nerve affection following diseases such as typhoid, dysentery, &c., need not be considered.

There is therefore no impossibility in the assumption that the disease is due to a poison manufactured in the body by a living organism which produces no constant visible lesions and no symptoms except the secondary effects due to the action of the poison these organisms have produced on selected nerve tracts.

Of the diseases mentioned, the two in which the causes are known are due to bacteria, diphtheria and tetanus. We have evidence that protozoal organisms, e.g., *Trypanosoma Gambiense* and *Spirochaeta pallida* in sleeping sickness and syphilis, can produce lesions of the nervous system, and some cases of paraplegia appear to be due to the parasites of malaria.

There are points in connection with the history of Beri-Beri that more closely resemble protozoal diseases than those due to bacterial action.

The most important of these is the great variation in the course of the disease, in the period of exposure required for the production of the disease, and especially the peculiar combination of tendency to relapse with acquirement of partial immunity. All these have close parallels in malaria in man and piroplasmosis in cattle.

Relapses take place in Beri-Beri frequently. They are most common at short intervals after the primary attack, but occur also after long intervals (*e.g.*, Taiping Gaol) when there is no possibility of reinfection, and may recur for a period of many years in persons who have suffered severely in a milder form.

On the other hand, newcomers suffer most severely. Without any alteration other than prolonged residence, the case-incidence diminishes in persons equally exposed from the beginning. In a gaol, the case-incidence is greatest amongst those inmates who have not previously had the disease in the gaol, whilst the long-sentence prisoners who have had attacks previously in gaol do not suffer severely, though they may have recurrences of symptoms to a mild extent. This was well shown in party No. 4, in Hamilton Wright's experiment.

No doubt many of the so-called relapses are in reality reinfections, as is shown by the great increase in "relapses" which accompanies a prison epidemic. The same rule holds true of the occurrence of malaria in a place with a high endemic index. In both diseases, if the possibility of fresh infections be excluded, the proportion of relapses, after considerable intervals free from any manifestation, is comparatively small.

Attempts at isolation of organisms from various parts of the body have been made. Like all other recent observers, in none of the organs, fluids, or tissues of the body normally free from organisms, have I been able to find parasites bacterial or protozoal. Cultures in various media in cases examined shortly after death have always remained sterile.

From any part of the alimentary tract, organisms in profusion are obtained as well as from the contents of the intestines. No constant deviation from the normal flora of these regions were detected. The recent increased knowledge of the minute forms of some protozoa and the additions made to our knowledge by recent advances, show that in yellow fever, hydrophobia, and other undoubtedly parasitic diseases, the parasites may not be found by ordinary methods.

It must be admitted that in spite of the large amount of work published as the result of observations in the Malay Peninsula by Simon, Ellis, Braddon, Travers, Wright, Durham, and other observers, as well as the large amount of unpublished observations made by the other medical men in the Peninsula, the cause of Beri-Beri is still to be determined.

We can claim that the issues are narrowing, and our knowledge more definite, but the nature of the true cause still awaits a discoverer. All I aim to do is to repeat and collect, with certain additional information, suggestions and ideas more or less distinctly formulated by others, and to direct attention to what I consider to be the most plausible directions for future work.

CONCLUSIONS.

1. That Beri-Beri is an infectious disease. As a rule, a short period of incubation and a period of exposure of less than three months is requisite for full development of the disease where the "endemic index" is high.

2. That there is no definite proof that an intermediate host is required, but the balance of evidence is against its being conveyed by earth, air, water or food, or contamination with sewage or other faecal matter.

3. That there is some evidence that for a short period only after the occupation of small spaces, beds, bedrooms, &c., the "poison" or carrier of infection may remain.

4. That food, either as regards quantity and quality, its nature or relative proportions, may have an effect on the susceptibility of the patients, though the proofs are not conclusive, but is not the causative agent.

5. That if an intermediate host for the unknown parasite is required, it must be either a cimex or a pediculus. That pediculi as carriers would better explain the incidence of the disease than any other blood-sucker.

6. That a closer enquiry into the earlier stages of the disease is required. That where opportunities for such an enquiry occur, renewed attention should be bestowed on the blood and tissues, with a view to determining the presence or absence of any protozoon.

7. Prophylaxis. That in view of the failures of various attempts at disinfection of buildings and places, and of various modifications of diet to have marked effects, more attention should be paid to limiting the chances of personal infection, and that particular attention should be paid to the personal cleanliness, freedom from vermin, and isolation of early or trivial cases of the disease.

The importance of the disease, affecting as it does the imported labour of the country, causing prolonged sickness and frequently death, cannot be over-estimated in a rich country so sparsely populated as the Federated Malay States. There is evidence that the disease is less common and less fatal than a few years ago; but directly, and as a complication of other diseases, it is still the main cause of the high mortality in the healthiest period of life, amongst the Chinese.

Every earnest endeavour to improve the conditions of life that has been made in the past, has resulted in an improvement. The number of cases is diminishing, and the mortality from the uncomplicated disease less than ever.

The scope of the enquiries has, as a result of the observations of numerous workers, been diminished, and the prospects of an early solution of the cause of the disease and of its mode of propagation may be confidently anticipated by future workers at no distant date.

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

- Vol. 1., No. 1.—The Malarial Fevers of British Malaya, by Hamilton Wright, M.D. (McGill.)
- Vol. 2., No. 1.—An Inquiry into the Etiology and Pathology of Beri-Beri, by Hamilton Wright, M.D. (McGill.)
- Vol. 2., No. 2.—On the Classification and Pathology of Beri-Beri, by Hamilton Wright, M.D. (McGill.)
- Vol. 3., Part 1.—The Diseases of British Malaya.
- Vol. 3., Part 2.—Water Supplies (Preliminary Observations).
- Vol. 3., Part 3.—The Culicidæ of British Malaya. (In the Press.)
- Vol. 3., Part 4.—The Outbreaks of Rinderpest in Selangor, 1903 and 1904, by C. W. Daniels, M.B. Camb.
- Vol. 4., Part 1.—Observations on Beri-Beri, by C. W. Daniels, M.B. Camb.
-

No. 1., Vol. 2 and 3, published by Kelly & Walsh, Singapore.
Vol. 2, Part 2, published by Bale, Sons & Danielson, London.

STUDIES
FROM
**THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.**

No. 9.

SURRA
IN THE FEDERATED MALAY STATES

BY
HENRY FRASER, M.D. (ABER.),
Director of the Institute for Medical Research

AND
S. L. SYMONDS, V.S. (MELB.),
Government Veterinary Surgeon.

WITH A NOTE
ON THE DISTRIBUTION OF CERTAIN SPECIES OF BITING FLIES
IN THE FEDERATED MALAY STATES

BY
H. C. PRATT.
Government Entomologist

Printed by Authority of the Resident-General, F.M.S.

Singapore:
KELLY & WALSH, LIMITED, PRINTERS,
SHANGHAI, HONGKONG & YOKOHAMA.

1908.

Price: Three Shillings net.

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

No. 9.

SURRA
IN THE FEDERATED MALAY STATES

BY
HENRY FRASER, M.D. (ABER.),
Director of the Institute for Medical Research

AND
S. L. SYMONDS, V.S. (MELB.),
Government Veterinary Surgeon.

WITH A NOTE
ON THE DISTRIBUTION OF CERTAIN SPECIES OF BITING FLIES
IN THE FEDERATED MALAY STATES

BY
H. C. PRATT.
Government Entomologist.

Printed by Authority of the Resident-General, F.M.S.

Singapore:
KELLY & WALSH, LIMITED, PRINTERS,
SHANGHAI, HONGKONG & YOKOHAMA.

1908.

Price: Three Shillings net.

to time
ments.
nment
a mare

MOIR,
er the

Gajah ;
Weld
e, and
ven of
were

uence

videly
esley,
and
erak.
hich
is an
onths
not
far as
was
than
were
ot be
ative;
atory

urred,
to be
alled
ound
were
bour

e led
tions
s in
they
have

ous districts of the States of Selangor and Negri Sembilan;
aded animals suspected of having the disease, animals known to

Surra in the Federated Malay States.

In the course of the past few years this disease has from time to time been observed in these States, as well as in the adjacent Straits Settlements.

In 1903 the first case was reported by Mr. FORD, Government Veterinary Surgeon, Selangor, the animal affected was an Australian mare at Klang and belonged to H. H. the Sultan of Selangor.

In 1905 an epidemic in the Krian district was investigated by Mr. MOIR, Government Veterinary Surgeon, Perak. From January to December the outbreak was responsible for the deaths of 43 ponies.

In August 1905 the disease occurred on Kellas Estate, Batu Gajah; sixteen bullocks had been imported from India and landed at Port Weld where they were quarantined; on release they were taken to this estate, and shortly afterwards, as reported by Dr. LEICESTER and Mr. FORD, eleven of the animals died from trypanosomiasis; in addition three ponies were found to have contracted the disease.

In September 1906 the disease recurred at Klang and in consequence this investigation was initiated.

During 1907 the disease occurred at various places which are widely apart; in horses it was met with in the Klang district, Province Wellesley, and Penang; in cattle it was met with in the States of Selangor and Negri Sembilan; Moir reported the disease amongst dogs at Taiping, Perak.

The first case observed by us was that of an Australian mare which was kept on Sungei Rengam Estate, Klang district; the estate is an isolated one and the mare had not been away from it for several months previously; two other horses, kept in the same stable, did not develop the disease. No other horses were kept in the vicinity and so far as could be ascertained no other horses had been near the estate. It was therefore deemed advisable to seek for some source of infection other than an equine one. The cattle on the estate and in the neighbourhood were examined; two animals were found whose emaciated condition could not be accounted for, and microscopical examination of their blood was negative; they were therefore tested by inoculation of their blood into two laboratory animals and both bullocks were thus proved to be infected.

At Klang town, 7 miles away from where the previous case occurred, in September two horses and in December one horse were discovered to be sufferers from the disease; four emaciated cattle which were found stalled near these horses were examined, in one trypanosomes were found microscopically on the sixth day of observation, and of the others two were proved, by the inoculation of their blood into laboratory animals, to harbour the parasites.

The apparent connection between the disease in horses and cattle led to a more extended investigation of the cattle; for this purpose inoculations of blood were made into laboratory animals from forty-five bullocks in various districts of the States of Selangor and Negri Sembilan; they included animals suspected of having the disease, animals known to have

been in contact with cases of the disease, as well as some apparently healthy animals. Fifteen bullocks were in this way proved to harbour the parasites; these cattle were either Indian, Siamese or cross-bred Indo-siamese. Three apparently healthy bullocks and one buffalo which were in the quarantine station at Port Swettenham were tested by animal inoculation, two of the bullocks proved to be infected. The animals had just been imported with a number of others from Kedah, one of the Siamese states. The animals tested were chosen at random, and there was nothing to suggest that they were sufferers from disease.

The Federated Malay States comprise the States of Perak, Selangor, Negri Sembilan and Pahang; they form a considerable part of the Malay Peninsula, certainly that part in which development has been greatest, especially in recent years. Adjacent to Negri Sembilan is the Straits Settlement of Malacca, and to the north of Perak is the Straits Settlement of Province Wellesley, these states are bounded on the north by the protected Siamese states of Kedah, Rahman, Ranga, Kelantan and Trengganu, which are of importance for this investigation not on account of their development, but from the fact that a number of cattle are brought from them or through them into Perak and to a less extent into Pahang. To the south of the Federated Malay States is the State of Johore; it is not a cattle raising country and is now in course of development.

From 2,000 to 3,000 horses and from 30,000 to 50,000 head of cattle are imported annually to Penang, Singapore, and the Federated Malay States. They are landed for the state of Perak at Prai, Port Weld and Telok Anson, for the state of Selangor at Port Swettenham, for Negri Sembilan at Port Dickson and Malacca; a very limited number enter Pahang by Kuantan on the east coast.

Once on the mainland their subsequent history cannot easily be followed as they pass from one part of the State to another and from State to State. The majority of these animals are slaughtered for use as food, the remainder are destined for transport purposes, as the principal mode of transport, apart from the railways, is by means of bullock carts. Year by year the mileage of roads increases, new estates and mines are constantly being opened up, and it is thus essential that an ever increasing number of transport animals should be imported, the natural increase being small and totally inadequate to meet the requirements.

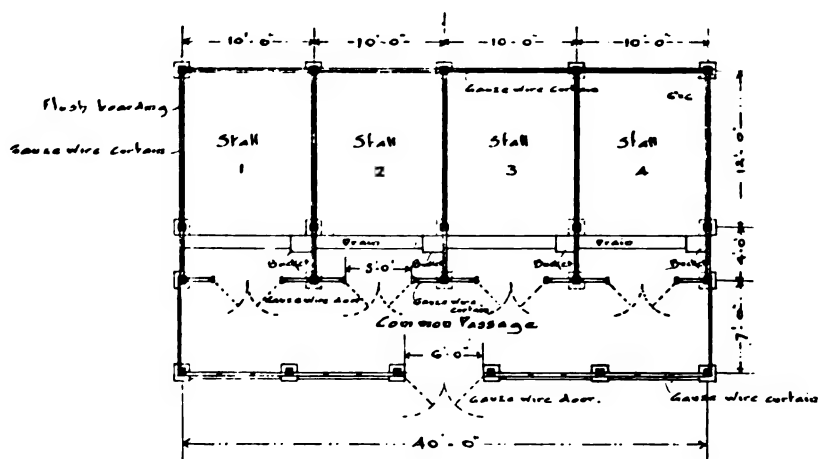
Cattle are brought from India by sea, and from Siam and the Siamese states overland as well as by sea. Horses and ponies are imported from Australia, Java and Sumatra.

In India, Siam and the Siamese states Surra is endemic. In Java and Sumatra the disease is known to occur. In Western Australia trypanosomiasis is stated to have occurred in 1907 among 500 camels imported from India. In the eastern states of Australia, from which most of the horses for this country are exported, the disease has not yet been observed.

Malay buffaloes (Kerbau) exist in considerable numbers in districts mainly peopled by Malays and are met with from time to time in other districts. In the Malay districts cultivation of paddy is the principal occupation and for tillage of the soil these animals are suitable. In forest



FLY-PROOF STABLE.



PLAN OF FLY-PROOF STABLE.

work they are most useful for the haulage of timber, but are too slow to be of use for transport purposes on metalled roads and being animals of a peculiar temperament can only be handled by their masters, the Malays; the consequence is that in places where the land has been opened up for rubber cultivation and tin mining the Malay buffalo has for all practical purposes disappeared. Microscopically we have not found trypanosomes in the blood of these animals, and we have not had an opportunity of making inoculation experiments from them.

Technique.

For the purposes of this investigation a four stalled fly-proof stable (photo and plan) was erected, the roof and walls were fitted with brass wire gauze, and the floor was of concrete. Each stall was completely isolated from its neighbour, the wire gauze being protected by a lining of wood which extended for over two-thirds of the height; the floor in each case sloped towards a metal bucket which was sunk in the floor and emptied daily; a moveable wooden floor was also provided. Running along the front of the stalls was a common passage likewise protected with wire gauze and drained independently of the stalls; double doors were fitted. Every effort in fact was made to secure isolation and to prevent the entrance of insects. Naturally infected horses and cattle were from time to time brought to the Institute and kept in this stable for purposes of observation and experiment.

The laboratory animals were kept in fly-proof cages.

All experimental inoculations were conducted so far as possible with aseptic precautions. Syringes with solid glass plungers were found most suitable for the removal of blood, which in the case of horses was taken from the jugular vein and in the case of cattle from one of the aural veins. The blood so obtained was at once inoculated into the experimental animal. No citrate solution or other diluting fluid was mixed with the blood; clotting in the syringe did not give trouble provided that the transference was performed without delay.

By the *incubation period* is to be understood the interval which elapses between the inoculation of infected blood and the microscopical demonstration of the parasites in the peripheral blood of the experimental animal.

Stained films were prepared by the use of Leishman's Stain, Giemsa's Stain, and the modification of Romanowsky's Stain introduced by Laveran and Mesnil.

The microscopical estimation of the richness of a blood in parasites was carried out by the examination of stained films. When a careful and prolonged search failed to find a single trypanosome they were stated to be absent; when only one or two were found in a film they were considered to be scanty; when they were rather more numerous they were estimated as few; when in almost every field examined several parasites were found they were described as plentiful; and when every field showed numerous parasites they were stated to be swarming. The method may not be one of great accuracy but when it is remembered that blood films from every animal were examined at least once daily, it must be admitted that our observations were sufficiently accurate for purposes of comparison.

Horses.

The course of the disease was followed in 7 horses which were infected by subcutaneous inoculation, and in one horse which was infected by means of biting flies. Observations have also been made on 7 naturally infected horses.

The period of incubation ranged from 4 to 9 days, the maximum period having occurred in the horse which was infected by means of biting flies.

The earliest clinical sign observed is a rise of temperature, which approximately coincides with the first detection of trypanosomes. The animal then appears listless, a feature which is more marked on exertion. After from two to five days the temperature returns to normal and the animal appears to improve. An afebrile interval of from one to four days is followed by a relapse, this in its turn is succeeded by an afebrile period and so on throughout the course of the disease; in the later stages of the disease the temperature may remain constantly elevated.

Emaciation is early noted and becomes accentuated as the disease progresses, the appetite however remains unimpaired even during an attack of fever.

Oedema of the legs first makes its appearance from two to ten days after the initial rise of temperature. In stallions and geldings oedema is next seen in the sheath and scrotum, which may become very much distended. In mares oedema of the mammæ occurs but it is not as a rule marked. Soon afterwards the oedema extends along the dependent parts of the abdomen and thorax. The amount of oedema varies in different cases, in some it may be extensive, in others it is only slight and varies from time to time. In two cases (a stallion and a gelding) oedema did not occur.

The pulse in the early stage is full; it is increased in frequency during the febrile periods and also at times in the afebrile intervals. As the disease advances the pulse becomes weaker and towards the end is usually intermittent; jugular pulsation may then be observed.

The respirations are but little increased in frequency during the early stages, later on they are accelerated and the acceleration may persist during the afebrile intervals. Dyspnoea may be present for a few days before death.

The conjunctival mucosa is at first congested, later it assumes a yellowish tinge and still later it becomes blanched; petechiæ have been observed. Lachrymation and a slight watery nasal discharge, which later on becomes mucoid, are frequently present.

Transient urticarial eruptions are not uncommon especially in the early stages of the disease; they occur on the forequarters and body.

When the disease has fully developed the emaciated animal presents a dull, listless, semi-somnolent appearance, it is disinclined to move, but when made to do so, staggers and there is evident weakness of the hindquarters.

From now onwards the progress of the disease is rapid, finally the animal falls and is unable to rise, in this position it may live for two or three days, or death may rapidly ensue.

The duration of the disease varies considerably, in the experimental cases it ranged from 8 to 78 days, and so far as could be ascertained in the naturally

infected cases it was from 60 to 80 days. Poor conditioned animals succumb more rapidly than those well conditioned and vigorous. In naturally infected animals the symptoms observed will vary with the stage at which the animal is brought under observation, in the initial phases or in the non-œdematous form the diagnosis may present some difficulty, but when the clinical signs are fully developed, the disease can easily be recognized. The demonstration of the trypanosome is requisite in order to clinch the diagnosis.

The number of parasites is, as a rule, proportional to the height of the temperature, thus with a high temperature they are abundant and with a normal or low temperature prolonged search may fail to detect them. The longest period during which trypanosomes could not be found was four days. At the time of death they may be swarming, few, scanty or not found (chart 1).

EXPERIMENT 306.

In order to follow the course of the disease in a poor conditioned horse an emaciated Australian mare, 5 years old, was inoculated subcutaneously with 2 c.c. of blood from an infected donkey (Exp. 286). Parasites were found on the 4th day and the temperature rose to 102°F, this was followed in 7 days by an afebrile period of one day, thereafter the temperature became remittent and so continued throughout the course of the disease. Parasites were as a rule found with ease; at one time they could not be seen for three days and at another time for one day. Oedema of the legs was first noted on the 6th day and by the 9th day it was pronounced; slight œdema occurred along the abdomen and thorax. On the 13th day weakness of the hindquarters was evident. On the 27th day the mare was found lying down, she was sweating profusely and the breathing was stertorous; death occurred a few hours later.

EXPERIMENT 343.

In order to follow the course of the disease in a horse inoculated from a naturally-infected bullock, an Australian mare, 9 years old, about 16 hands high and in excellent condition, was inoculated subcutaneously with 1 c.c. of blood from a naturally infected bullock (No. 51.) Microscopically trypanosomes could not then be found in the blood of the bullock. Seven days later the temperature of the horse rose to 103°F and on the following morning trypanosomes were discovered. Thereafter the temperature was irregular, a remittent temperature of from two to five days duration alternated with an interval of one to two days, when the temperature was either intermittent or normal. During the last 12 days of the disease the temperature remained constantly elevated.

On six occasions parasites could not be found; their period of absence lasting from one to four days.

Oedema of the legs was first noted on the 10th day and by the 13th day was marked; on the 18th day œdema under the abdomen was observed and later on it extended along the thorax.

An urticarial eruption occurred from time to time.

Loss of condition was pronounced by the 25th day and weakness of the hindquarters, which was first noted on the 44th day, became very evident by the 50th day.

On the 62nd day the mare fell down and did not rise again. Death occurred on the 66th day.

EXPERIMENT 384. (*Chart I*).

It is of interest to compare this case with the previous one. An Australian gelding, 8 years old, in good condition was inoculated subcutaneously with 0.1 c.c. of blood from a naturally infected bullock (No. 63). The period of incubation was six days and the disease ran its course in 51 days. The observations as regards parasites and clinical signs were similar to that in the preceding case, save that oedema was never present. That this was not due to a different species of trypanosome was shown by the fact that another horse which was inoculated with the same strain of trypanosomes developed oedema of the legs, abdomen and thorax.

EXPERIMENT 280.

In order to note the course of the disease in a horse infected by means of biting flies two flies of the genus *Tabanus* were allowed to feed on a horse (Exp. 343.) in whose blood trypanosomes were then plentiful. The flies were immediately afterwards transferred to and fed on an Australian gelding, 9 years old, and in good condition. On the 9th day trypanosomes were found.

The clinical signs and the occurrence of the parasites were similar to those noted in Exp. 343. Death occurred on the 78th day.

EXPERIMENT 434.

In order to note the course of the disease in a horse inoculated from a naturally infected dog, an Australian gelding in very poor condition was inoculated with 0.25 c.c. of blood from dog (431). Six days later trypanosomes were found in the blood of the horse and on the 8th day death occurred. The parasites were then swarming.

Post-mortem Appearances in Horses.

There are no lesions characteristic of this disease. Post-mortem rigidity occurs early and is usually well marked. In oedematous cases a yellowish gelatinous material infiltrated the subcutaneous tissues of the dependent parts of the abdomen and thorax.

The pleural cavities did not usually contain an excess of fluid. The lungs, apart from the presence of a few petechiæ, were normal.

The pericardial sac contained a variable amount of serous fluid. The heart did not show any increase in size; around its base and along the interventricular groove there was frequently found a quantity of gelatinous-like material which had replaced the fat. Clots were always met with in the right auricle and ventricle, but might also be found in the left auricles and ventricle. When death had been lingering they were most extensive and then occurred as pale yellow or orange coloured masses which practically filled all the cavities of the heart and extended into the great vessels; Petechiæ both epicardial and endocardial have been noted.

In the œdematous cases a variable amount of clear straw-coloured serous fluid was found in the peritoneal cavity.

The liver, kidneys and suprarenal bodies showed nothing abnormal.

The spleen was in some cases enlarged and might show some petechiæ, the pulp was apparently normal.

The stomach, intestines and bladder as also the brain and spinal cord presented no macroscopical lesions.

Cattle.

The disease may apparently manifest itself in epidemic form, as reported in the outbreak on Kellas Estate; we have not, had the opportunity of investigating such an outbreak.

In all 30 cases have been studied; of these 24 were naturally infected animals, and 6 were infected by subcutaneous inoculation. They included Indian, Siamese and cross-bred Indo-siamese cattle; the naturally infected animals were found in various districts and places often widely apart.

The course of the disease in cattle is apparently dependent on the condition of the animal at the time of infection.

Animals, in good health who have become infected, may show no interference with their condition, apart from an occasional rise of temperature. On the expiry of the incubation period parasites are found in the peripheral blood, they may remain present for some days, and continue to be found at intervals, ultimately they disappear entirely and after several months the inoculation of their blood, even in large quantities into susceptible animals fails to produce trypanosomiasis. The ultimate fate of such cattle is difficult to determine, recovery may occur, but in a country such as this where the disease is endemic it is possible for such animals to become reinfected on one or more occasions, and their health eventually may be interfered with. One attack of the disease does not necessarily confer immunity. On the other hand, they may be attacked by an intercurrent disease, as for example foot and mouth disease, or become run down as the result of overwork, trypanosomiasis may then reassert itself, and death ultimately occur from the original and not the intercurrent disease.

When animals in poor condition are attacked, the disease usually runs a course which is more acute

The incubation period ranged from 4 to 7 days. Approximately coincident with the occurrence of trypanosomes, the temperature rises and its subsequent course varies considerably in different cases; as a rule febrile intervals of a few days' duration alternate with afebrile intervals which show great variations in their extent; even in well advanced cases of the disease a normal temperature may exist for several weeks in succession. In other cases the temperature is irregular.

In naturally-infected animals kept under observation until a fatal result ensued, exacerbations of temperature are more frequent towards the end. In animals which apparently recover, the temperature is irregular for about six months after the last occasion on which the parasites are found microscopically

When the blood ceases to be infective for susceptible animals the temperature becomes normal. The clinical signs are indefinite, when there

is obvious interference with health progressive emaciation of the affected animal is the rule, so that it becomes less and less fit for work. The appetite is at no time interfered with during the course of the disease and the bowels perform their excretory functions normally.

Oedema has never been observed, nor has there been any glandular enlargement.

The coat loses its glossy appearance, and the hair becomes scanty in parts. In some instances eczematous eruptions are noted, they extend over the head, neck, forequarters, and along the back; when healing occurs the parts, which have been affected, remain denuded of hair.

Lachrymation and a mucoid nasal discharge are at times noted; the visible mucous membranes ultimately become markedly anæmic.

The respiratory and cardio-vascular systems present nothing of note.

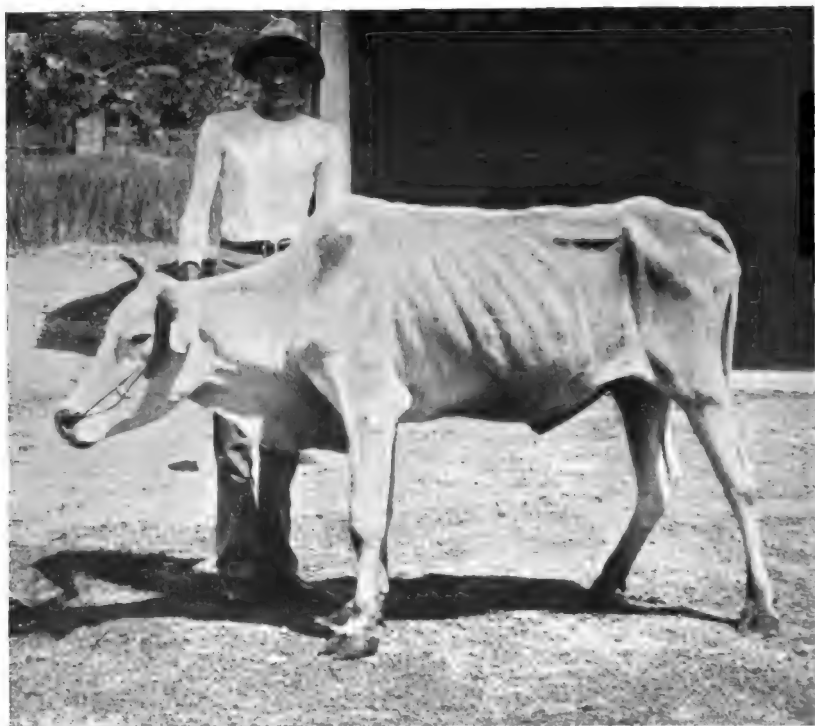
As the end approaches the emaciated animal spends most of its time lying down, ultimately it becomes too weak to rise and after a few days in the recumbent posture death quietly ensues.

The duration of the disease cannot be stated with any degree of accuracy, naturally infected animals were usually stated to have been ill for some six or eight months prior to their coming under observation, and their death has occurred from two days to four months later. In the case of cattle experimentally infected and in which a fatal result occurred, the disease ran its course in periods which ranged from 17 days to six months. If cases be considered in which apparent recovery took place, accuracy is impossible, observations of temperature are insufficient microscopical examination of the blood will not answer, and the inoculation of the blood into laboratory animals is not necessarily an infallible test.

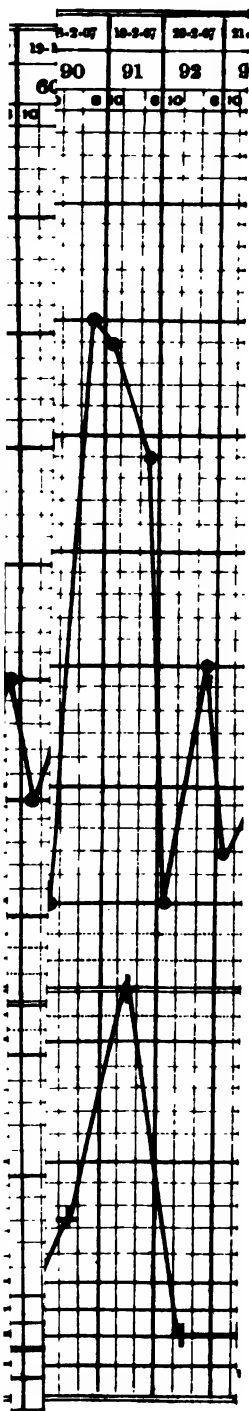
As regards the occurrence of the parasites, they will be found microscopically as already stated on the expiry of the incubation period (Chart III.) and may then persist for several days; their subsequent occurrence is dependent on the condition of the inoculated animal. In the case of healthy cattle they may appear on several occasions, but later tend to remain absent for long periods; in one case after an interval of absence of 45 days, they reappeared for one day, there-after they could not be found; in another case after 3 months' absence they reappeared in large numbers. In the case of ill-conditioned cattle parasites will more frequently be found.

In the initial stages of the disease and towards its fatal termination, there is usually some relationship between the number of parasites present and the temperature but in the intermediate stages parasites may be present during an afebrile period and absent during a febrile one. In the microscopic examination of blood from infected cattle parasites are either found with comparative ease or are not to be discovered by a prolonged search and in this respect they differ from infected horses in whom a careful search will often reveal at least one trypanosome.

Generally speaking the detection of trypanosomes in cattle is not an easy matter, no doubt cases are met with in which parasites are found on the first examination or after the animal's blood has been examined for some days in succession, but as a rule the inoculation of the suspected blood into an animal, such as a guinea-pig, is essential to expedite the diagnosis.



NATURALLY-INFECTED SIAMESE BULLOCK (No. 51).



Naturally Infected Cattle.

The following two cases have been selected as examples of the history and course of the disease in cattle.

(No. 51.) (*Photo. 2. Chart II.*)

An aged Siamese bullock was proved by animal inoculation to be infected; the animal was in very poor condition and unfit for work, there was an eczematous eruption over the forequarters and back.

The bullock was stated to have been worked in Selangor for a number of years; six to eight months ago the animal began to lose condition and was then only worked occasionally. It was brought to the Institute 11 days after it was first seen.

Under observation the temperature remained practically normal for about three weeks; a febrile interval of short duration then occurred and was followed by an apyrexial interval, thereafter to be succeeded by frequent pyretic attacks of gradually increasing duration.

Parasites were found microscopically on the 13th day, after which they could not be found until the 48th day, they then reappeared at intervals practically coinciding with the occurrence of fever. During the last month of the illness trypanosomes were found every few days and on these occasions they persisted for from two to three days. At the time of death, which occurred after the animal had been under observation for four months, parasites were extremely abundant.

(No. 63.)

A cross-bred Indo-siamese bullock was proved to be infected by animal inoculation. The bullock was born in Kuala Lumpur in 1900, since 1904 it had been worked regularly in that town. Five months previous to its coming under observation it was taken to Negri Sembilan where it contracted foot and mouth disease; it was quarantined with other affected animals, since then the bullock had gradually become thinner and was in consequence only worked occasionally.

When the animal first came under notice it was in poor condition and had a dull listless appearance, the hair was scanty in parts but there was no eczema. The bullock was brought to the Institute where it died after 17 days. Trypanosomes were on one occasion present for three consecutive days, the temperature then ranged from 102° to 103°F. At the time of death parasites could not be found.

Experimentally Infected Cattle.

EXPERIMENT 318. (*Chart III.*)

In order to note the course of the disease in a perfectly healthy animal, a Siamese cow about 3½ years old and which had been found free from the disease by animal inoculation, received subcutaneously 5 c.c. of blood from a horse in which the parasites were then swarming.

On the evening of the fourth day the temperature rose to 104° F. and on the following morning parasites were found microscopically; the temperature then fell to normal, but rose again in the evening to 105° and

remained elevated for two days, thereafter it became intermittent. At intervals there were febrile attacks of from one to two days' duration but after six months the temperature became normal and remained so.

The parasites after their first appearance remained present for three days but were never abundant; they then disappeared for a day and returned for two days, they were not again found until the 51st day; from that day routine daily examination failed to detect the presence of trypanosomes. On the 162nd day, by animal inoculation, parasites were proved to be present in the peripheral blood. On the 267th day, a rabbit was inoculated with 0.5 c.c. of the blood but did not become affected, and on the 310th day a rabbit and a goat were each inoculated with 10 c.c. of the blood but neither of them became affected.

During the first few months the cow lost condition but afterwards regained apparently perfect health.

On the 233rd day she was delivered of a well developed calf and successfully reared it.

Ten and a half months after the first inoculation the cow was inoculated with 10 c.c. of blood from a naturally infected pony in whose blood trypanosomes were then swarming.

On the 6th day parasites were found in the blood of the cow and persisted for three days, they then disappeared for three months after which they reappeared in great numbers for two days, thereafter they could not be found. The temperature was very irregular and the animal became much emaciated; now, seventeen months after the first and six months after the second inoculation, the animal is still alive.

EXPERIMENT 435.

The calf of the previous animal was proved free from trypanosomes by animal inoculation and when two months old it was inoculated with 10 c.c. of bloody serum taken from the heart of a bullock at a post-mortem performed fourteen hours after death.

Fourteen days later trypanosomes were found in the peripheral blood, they remained present for three days, the examination was continued for six months with negative results.

The temperature chart at times showed some irregularity. The calf never appeared to suffer in any way and is now in excellent condition.

EXPERIMENT 386.

In order to note the course of the disease in an animal in poor condition, an aged, emaciated Siamese bullock was proved free from trypanosomes by animal inoculation and thereafter received subcutaneously 5 c.c. of blood from a horse (Experiment 384) in whose blood parasites were then scanty.

On the eleventh day trypanosomes were found in the peripheral blood, they remained present for four days, subsequently they were found on three occasions of from one to four days' duration.

The temperature was usually normal in the morning and elevated in the evening to 103° F. or thereabout.

A thin mucoid nasal discharge occurred, and about a month after inoculation a dry scaly eruption appeared on the neck and forequarters.

Emaciation became more and more marked and the animal passed most of its time lying down. Death occurred on the 78th day, trypanosomes were then absent.

EXPERIMENT 405.

On this occasion an emaciated Indian bullock was employed, it was proved free from trypanosomes by animal inoculation, and then received 0.25 c.c. of blood from a rabbit which had been inoculated from a naturally infected bullock. On the 7th day trypanosomes were found in the peripheral blood, they were then scanty, but they gradually increased in number until on the 13th day they were swarming, they next disappeared for two days, on the 16th day they were again swarming, as also on the following day in the evening of which the animal died. After the onset of the disease the bullock became very dull, it remained lying down most of the time, but the day before death it became brighter, and a few hours previous to that event it was standing up and eating. The temperature throughout ranged from 102° to 103° F.

Post Mortem Appearances in Cattle.

There are no characteristic pathological changes. No subcutaneous infiltration was observed in any case.

The pleural cavities did not contain an excess of fluid and the lungs were normal. The quantity of pericardial fluid was usually somewhat increased, but overdilatation was never noted. The heart was not enlarged, sometimes it was found in a state of systole and its cavities empty, but more usually these contained varying amounts of clots, especially common and marked in the right ventricle. The large ante-mortem or "goose-fat" clots observed in the heart and at times in the large vessels of horses were never seen in cattle. Subendocardial hæmorrhages were not uncommon.

The peritoneal cavity generally contained a moderate amount of clear serous fluid. The spleen was never enlarged, petechiæ were frequently present, the pulp appeared normal. The other abdominal viscera were healthy; the brain and spinal cord showed no macroscopical lesions.

Dogs.

We have not found dogs naturally infected with trypanosomes but Moir of Perak has observed seven such cases. The symptoms noted by him were intermittent fever, loss of appetite, disinclination for exertion, rapid emaciation, and in one case opacity of the cornea. One of these dogs was sent to the Institute, and from it various inoculation experiments were made. These experiments as well as morphological comparison of the trypanosomes with those from bovine and equine sources, have shown that the parasites obtained from naturally infected horses, cattle and dogs are indistinguishable.

EXPERIMENT 431.

A pariah dog in good condition was inoculated subcutaneously with 2 c.c. of the heart's blood taken from the Perak dog. Five days later parasites were found in the peripheral blood, thereafter the parasites were constantly to be found and for three days previous to death, which occurred on the thirty-fourth day, they were swarming. The temperature continued

elevated after the onset of the disease. By the 8th day emaciation was evident and rapidly became more marked. The dog then appeared dull and listless, corneal ulcers developed in both eyes, and towards the end there was paresis of the hindquarters.

An autopsy was performed 18 hours after death. The pericardial sac and peritoneal cavity contained a quantity of blood-stained fluid. All the cavities of the heart contained post-mortem clots, that in the right ventricle being small. The spleen was much enlarged and the pulp was soft and friable. The lymphatic glands throughout the body were enlarged and congested. The other viscera were apparently normal.

EXPERIMENT 385.

Several biting flies (*Tabanus minimus*) were allowed to feed on an infected horse (Exp. 384) and immediately afterwards transferred to a Chinese hairless dog on which in some cases they resumed their meal, 7 days after the last biting fly had fed on the dog trypanosomes were found in the peripheral blood of that animal.

Two days later the dog appeared dull and depressed. On the following day clonic spasms were observed and two days afterwards death occurred preceded by epileptiform convulsions.

The temperature was irregular and elevated, although not so much so as in the previous case.

The results of the post-mortem were similar to those met with in the case 431.

EXPERIMENT 400.

A pariah dog was inoculated with 1.5 c.c. of blood containing trypanosomes of bullock origin. On the 5th day the parasites were found in the peripheral blood and six days later death occurred, at which time trypanosomes swarmed. The clinical symptoms and post-mortem results resembled those in the previous case.

Rabbits.

Eight rabbits were infected with trypanosomes which had been obtained from horses and nine with those obtained from cattle.

The incubation period was shorter in animals infected with horse trypanosomes than in those infected with cattle trypanosomes, but as will be seen from the tables the number of parasites inoculated was almost invariably greater in the former than in the latter animals.

Considerable variations were noted in the duration of the disease and it is scarcely possible to compare the two groups. Variations in virulence of different strains of trypanosomes, as also the condition of the experimental animal would complicate such a comparison.

In experiments 350 and 357 the biting flies were allowed to feed on the infected animal and then transferred to the rabbits on whom they resumed their feed, in both rabbits the period of incubation was 8 days and the duration of the disease was 49 days. Experiment 377 in which the rabbit was inoculated by the prick of an infected hypodermic needle, the period of incubation was 8 days and the duration of the disease was 44 days. The proboscis of the biting flies appears to act like a hypodermic needle, and thus the amount of inoculated material was in these three instances approximately comparable.

The temperature in these experimental animals is elevated and irregular.

Emaciation is the rule save in those cases in which the course of the disease is a short one.

Slight œdema of the genitals was occasionally noted, also loss of hair.

Conjunctivitis was commonly observed.

Trypanosomes after their first appearance in the peripheral blood increase considerably in number, and then gradually disappear to reappear after an interval of from 3 to 4 days, these waves continue throughout the disease. The time during which parasites persisted in the peripheral blood ranged from 1 to 14 days. At the time of death or just previous to that event they were absent in four cases, scanty in three, few in four, plentiful in three and swarming in three.

The time during which parasites could not be found ranged from 1 to 6 days.

Post-mortem. There are no characteristic lesions. The pericardial sac and the peritoneal cavity may contain some excess of fluid. The heart, especially the right ventricle may contain post-mortem clots. The spleen is never enlarged.

RABBITS.

| Experimental No. | Strain of Trypanosome. | Animal from which inoculated. | Amount of blood inoculated. | Microscopical estimation of the richness of the blood in parasites. | Period of incubation. | Duration of the disease. | REMARKS. |
|------------------|------------------------|-------------------------------|-----------------------------|---|-----------------------|--------------------------|---|
| 281 | Horse | Horse | 1 c.c. | Few | 3 days | 16 days | |
| 284 | " | " | 3 c.c. | " | 3 " | 23 " | |
| 288 | " | " | 3 c.c. | Plentiful | 3 " | 7 " | |
| 294 | " | Donkey | 2 c.c. | Scanty | 5 " | 12 " | |
| 301 | " | " | 2 c.c. | Few | 3 " | 5 " | |
| 339 | " | " | 0.1 c.c. | Scanty | 5 " | 9 " | |
| 360 | " | Bullock | 3 c.c. | Absent | 10 " | 28 " | |
| 426 | " | Horse | 1 c.c. | Plentiful | 5 " | 10 " | Killed for the examination of smears from the organs. |
| 304 | Bullock | Bullock | 3 c.c. | Absent | 8 " | 10 " | |
| 309 | " | " | 0.5 c.c. | " | 7 " | 39 " | |
| 324 | " | " | 2.0 c.c. | " | 7 " | 10 " | |
| 350 | " | Bullock | Tabanus Expt. | Scanty | 8 " | 49 " | Direct transmission. |
| 357 | " | Horse | " | Plentiful | 8 " | 49 " | Direct transmission. |
| 359 | " | Bullock | 2 c.c. | Absent | 8 " | 39 " | |
| 377 | " | " | Pricked with needle. | Few | 8 " | 44 " | |
| 380 | " | " | 2 c.c. | Absent | 8 " | 24 " | Killed for the examination of smears from organs. |
| 416 | " | Guinea-pig | 0.5 c.c. | Plentiful | 10 " | 40 " | |

Guinea-Pigs.

Four guinea-pigs were infected with trypanosomes which were of horse origin and ten with trypanosomes which were of bullock origin and one was infected with the parasites obtained from a dog.

The incubation period was on the whole shorter in the case of those animals infected with trypanosomes of horse origin than in those infected with trypanosomes of cattle origin; the explanation is the same as that given for rabbits.

The duration of the disease varied considerably and, as was the case in rabbits, comparison is scarcely possible.

In Experiments 382 and 389 which were made with biting flies the incubation periods were respectively 16 and 10 days, and the duration of the disease was 74 and 58 days. These may be compared with Experiment 305 where the incubation period was 6 days and the duration of the disease 62 days, also with Experiments 310 and 325 in which the number of trypanosomes inoculated was relatively small.

The temperature throughout the disease was irregular, there were no distinct febrile periods. Emaciation was observed in those cases in which the disease was of considerable duration, but apart from this there were no clinical signs. Parasites having once made their appearance tended to persist for longer periods than was the case in rabbits. They have been noted to remain present for 38 successive days; it was exceptional to be unable to find them for more than five or six days at a time although on one occasion they were absent for eleven days. When present they are usually abundant.

At the time of death or just previous to that event they were plentiful in six cases and swarming in nine.

Post-mortem. There are no characteristic lesions. The spleen never showed any marked enlargement.

GUINEA-PIGS.

| Experimental No. | Strain of Trypanosome. | Animal from which inoculated. | Amount of blood inoculated. | Microscopical estimation of the richness of the blood in parasites. | Period of incubation. | Duration of the disease. | Remarks. |
|------------------|------------------------|-------------------------------|---|---|-----------------------|--------------------------|---|
| 283 | Horse | Horse | 2 c.c. | Few | 5 days | 34 days | Inoculated with blood drawn 3 hours after death. |
| 289 | " | " | 2 c.c. | Plentiful | 5 " | 35 " | |
| 295 | " | Donkey | 2 c.c. | Scanty | 7 " | 8 " | |
| 300 | " | Horse | 2 c.c. | Few | 4 " | 25 " | |
| 305 | Bullock | Bullock | 2 c.c. | Absent | 6 " | 62 " | |
| 308 | " | " | 0.25 c.c. | " | 9 " | 21 " | |
| 310 | " | " | 0.5 c.c. | " | 11 " | 21 " | |
| 316 | " | Rabbit | 0.5 c.c. | Scanty | 15 " | 53 " | |
| 323 | " | Bullock | 0.5 c.c. | Absent | 7 " | 25 " | |
| 325 | " | " | 0.5 c.c. | " | 12 " | 37 " | |
| 362 | " | Rabbit | 0.25 c.c. | Few | 6 " | 18 " | Inoculated with blood drawn 3 hours after death. |
| 430 | Dog | Dog | 2 c.c. | " | 4 " | 36 " | Inoculated with blood drawn 4 hours after death. |
| 382 | Bullock | Bullock | Contents of Red Tabanus after 24 hours. | Absent | 16 " | 74 " | Contents of fly were shelled out and inoculated into pocket in groin of guinea-pig. |
| 389 | " | Horse | Contents of Red Tabanus after 24 hours. | Few | 10 " | 58 " | Contents of fly were shelled out. Cut up in salt solution and injected intraperitoneally. |

Rats.

The blood of these animals was invariably examined for some time previous to inoculation in order to determine the absence of *Trypanosoma Lewisi*, although morphologically it is scarcely possible to confuse this parasite with the *Trypanosoma Evansi* it was desirable that only normal rats should be employed.

Five rats were inoculated with trypanosomes of horse origin and four with trypanosomes of bullock origin. The incubation period in the former group varied from one to six days and in the latter group from one to five days.

The duration of the disease ranged from three to six days in the former group and from two to four days in the latter. No difference was observed in the course of the disease in either group. There are no clinical symptoms. The parasites having once appeared, steadily increased in number until towards the end, and at the time of death they were swarming.

Post-mortem enlargement of the spleen was noted in most cases; there were no other apparent lesions.

RATS.

| Experimental No. | Strain of Trypanosome | Animal from which inoculated. | Amount of blood inoculated. | Microscopical estimation of the richness of the inoculated blood in parasites. | Period of Incubation. | Duration of the disease. | Remarks. |
|------------------|-----------------------|-------------------------------|-----------------------------|--|-----------------------|--------------------------|------------------------------------|
| 282
White Rat | Horse | Horse | 2 c. c. | Few | 2 days | 6 days | |
| 290
Brown Rat | " | " | 1 c. c. | Plentiful | 3 " | 5 " | |
| 296
Brown Rat | " | Donkey | 1 c. c. | Scanty | 6 " | 4 " | |
| 298
Brown Rat | " | Rat | 0.25 c. c. | Swarming | 2 " | 3 " | Blood removed 2 hours after death. |
| 302
Brown Rat | " | Horse | 1 c. c. | Few | 3 " | 3 " | |
| 392
White Rat | Bullock | " | 2 c. c. | " | 2 " | 3 " | |
| 402
Brown Rat | " | " | 0.5 c. c. | " | 3 " | 3 " | |
| 403
Brown Rat | " | " | 0.25 c. c. | Plentiful | 3 " | 4 " | |
| 412
Brown Rat | " | Guinea-pig | Needle prick | " | 5 " | 2 " | |

Donkey.

A donkey (Exp. 286) about 2 years old and in good condition was inoculated with 6 c.c. of blood from a naturally infected horse. Six days later trypanosomes were found in the peripheral blood, the temperature was then normal and continued so for three days, after which it rose and remained elevated for three days. From then onwards afebrile intervals of from one to eight days alternated with febrile attacks of one to three days' duration, for four days previous to death the temperature was subnormal.

Emaciation gradually set in, and the animal became anæmic, œdema was never noted. No other clinical signs were observed. Four days previous to death the animal fell down, it did not rise again and died quietly on the 116th day.

The trypanosomes were usually most abundant when the temperature was elevated, they were absent on 12 occasions for periods which ranged from one to four days.

An autopsy was performed six hours after death. No lesions were observed, the heart was in a state of systole and its cavities empty.

Monkey.

A monkey (*Macacus nemestrinus*) (Exp. 297) was inoculated with 2 c.c. of blood drawn from the donkey (Exp. 286), after four days parasites were found in the peripheral blood, two days later the temperature rose and remained more or less constantly elevated throughout the course of the disease. The monkey gradually became anæmic and emaciated; during the last ten days of the illness the animal was generally asleep; it would fall asleep whilst eating, then wake up suddenly and resume the process. Rigors were frequent for four days previous to death which occurred on the 100th day.

Trypanosomes, after their first appearance remained present for 33 days, they were then not found for three days. They were never absent for more than four days and when present were usually found in fair abundance, just before death they swarmed, they were at that time found in the cerebro-spinal fluid.

A post-mortem examination was held just after death. There was no subcutaneous infiltration. The pericardial sac contained a moderate amount of clear serous fluid. The heart was in a state of diastole and its cavities were filled with blood.

The spleen was much enlarged, on section the pulp was found to be firm and of a dark red colour. The upper surface of the liver was covered with a yellowish-grey pseudo-membrane but that organ on section appeared normal.

The other organs showed no macroscopical lesions.

Goats.

(Exp. 398).

A Bengal goat in good condition was inoculated with 0.1 c.c. of blood containing trypanosomes of bullock origin. On the 8th day parasites were found in the peripheral blood, they were again found on the following day, they then disappeared for five days and returned for two days after which they could not be found.

The temperature rose on the fifth day and thereafter remained almost constantly elevated.

The animal gradually became emaciated but oedema was never observed, death occurred on the 47th day.

Post-mortem. There was nothing of note.

(Exp. 387).

A Bengal goat was inoculated with 4 c.c. of blood which contained trypanosomes of bullock origin. On the 6th day parasites were found and on the 70th day the animal died. Trypanosomes were found on the one day only, they then disappeared and were never met with again. The course of the disease was otherwise similar to the preceding case.

Biology and Morphology of the Trypanosomes.

In fresh blood the parasites exhibit very active movements, when kept for some time the activity lessens, and it can then be made out that the trypanosome usually moves with the flagellated extremity foremost; there is in addition a considerable amount of wave like motion in the body of the parasite, apparently originating in the undulating membrane, this is very noticeable when the passage of the parasite is retarded, as for example by a mass of blood corpuscles. Details of structure cannot be made out in such preparations, the parasites usually die in a few hours, but they have been found alive after 12 to 24 hours in preparations carefully sealed; such films kept at 22° C. did not show any appreciable prolongation in the life of the trypanosomes and the addition of citrate solution was of no benefit.

In stained preparations the structure can be made out and the different phases of division can be studied.

There is considerable range in the size of the parasites, the smallest one was observed in a film from the liver of a dog and measured 13m.* in length, inclusive of the free portion of the flagellum, and 1.5m. in its broadest part. In the peripheral blood they have been observed to range from 18m. to 34m. in length. The smallest of them measure at their broadest part 1.5m. to 2.7m. and the largest measure from 3m. to 3.4m. All possible gradations have been noted between these extremes of size. The breadth of the parasite is mainly dependent on the extent to which the undulating membrane has developed, in consequence short broad parasites may be found which markedly contrast with long slender ones. Dividing forms show distinct though less pronounced variations in size, the length ranged from 25m. to 34m. but the greatest breadth was almost invariably 3.5m. In forms which have just divided, the two separate parasites were of equal size, and measured from 20m. to 30m. in length, their maximum breadth did not exceed 2m. The length of the parasite was not less in rats, guinea-pigs and dogs than in horses and donkeys, contrary to the observations of Laveran and Mesnil.

The cytoplasm stains a light blue, granules may be observed in it. Vacuoles are not common, but when present they may be seen in front of the centrosome, and if large they distend the body of the parasite laterally at that place. The anterior extremity of the body is pointed but the posterior extremity varies, it has most commonly the form of an elongated cone, but may be blunt and rounded.

* For m read microns throughout.

1.



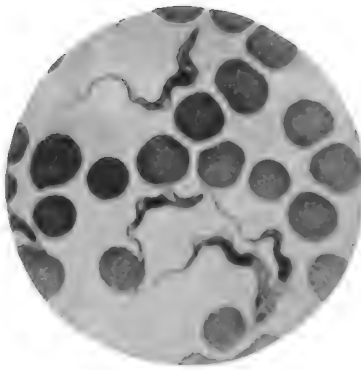
Trypanosomes from naturally infected horse.
x 1050

2.



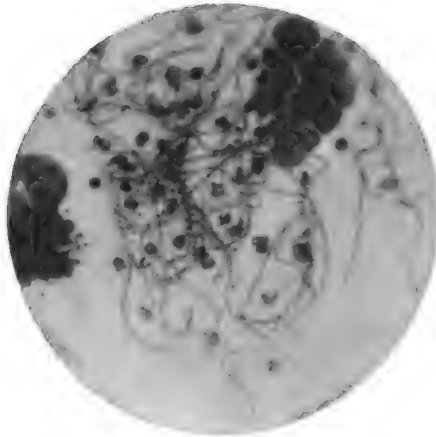
Trypanosomes from naturally infected bullock.
x 1050

8.



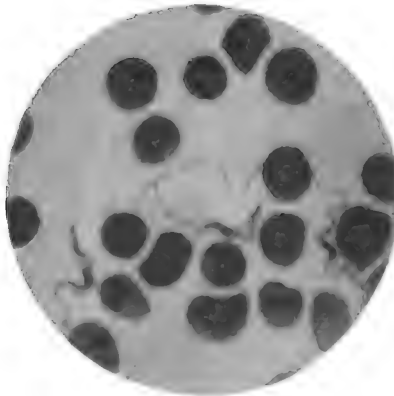
Trypanosomes from naturally infected bullock,
shows dividing form. $\times 1050$

4.



Involution forms from heart's blood, post-mortem
on guinea-pig (Exp. 283). $\times 920$

5.



Free flagellum in peripheral blood of
guinea-pig (Exp. 300). $\times 920$

The nucleus is situated at approximately the middle of the body, and is oval in shape, its length varied from 1.5m. to 5m., on an average 3m.

The centrosome in the pointed forms is situated from 1m. to 3m. from the posterior extremity; in the blunt forms it is close to the end.

The flagellum arises from this body, it passes forwards following the margin of the undulating membrane, whose folds it renders more prominent, and terminates anteriorly in the free portion which varies from 4m. to 10m. in length. The undulating membrane stains lightly and its somewhat hyaline appearance sharply demarcates it from the cytoplasm.

When the parasites are in process of division the centrosome is seen to elongate and then divide into two, the daughters separate some distance from each other in the long axis of the parasite and from each centrosome a flagellum is observed to take its origin more or less directly; proceeding forward they are seen to merge into one, later on the division becomes complete and two flagella as well as two centrosomes are present. By the time these changes are affected, the nucleus has divided and the appearance noted is that of two parasites in intimate contact. Separation occurs from the anterior part first, and the parasites swing apart usually into one long line, remaining connected by their posterior extremities: finally detachment is completed. The parasites which have just separated are not small, their size is dependent on that of the parent, each being approximately equal to it in length, although less in breadth. Binary division only has been observed.

Differentiation of the parasites into sexual and asexual forms, based on characteristics such as those given by Ziemann, Prowazek and others, could not be made out: by insensible gradations the one form passes into the other so that sharp lines of demarcation were impossible.

Moist preparations and stained films were examined from the heart's blood and various organs at 68 post-mortems. These observations were made at different intervals after death, the longest interval being 48 hours. Fully formed motile trypanosomes have been found 22 hours after death, but as a rule 12 to 15 hours after death no motile parasites can be seen and the trypanosomes show various stages of involution.

If at the time of death trypanosomes were plentiful, then the parasites were found in every organ, provided that the examination was made immediately after death; on the other hand, if at the time of death trypanosomes were not found in the peripheral blood, the most careful examination failed to reveal the presence of the parasites in any organ and we have not been able to recognize anything which could pass for an encysted or evolution stage of the parasite.

In the heart's blood trypanosomes were usually present in numbers corresponding with that met with in the peripheral blood, and the various stages of involution could be followed. The flagellum becomes released from the parasite, detachment finally occurring at the posterior extremity. The parasite meanwhile may remain elongated but usually tends to become somewhat rounded, the protoplasm gradually melts away and the nucleus becomes more prominent. The blepharoplast remains for a time in the neighbourhood of the flagellum but is ultimately lost sight of. We have then the flagellum removed to a varying distance from the nucleus which may be surrounded by a faintly stained halo of protoplasm. The detached

flagella have on occasions been observed to be thickened (Fig. 4) but as a rule they appear to have the same size as in the normal parasites. The nuclei are the most persistent structures and in their free state show as it were bipolar staining, the chromatic substance is aggregated into two masses, a clear faintly staining area intervenes between them.

Great clumps of these involution forms have from time to time been noted and the suggestion is forced on one that they may have been a contributory cause, if not the cause of death. When trypanosomes are present in abundance, various involution forms can be seen, these if not rapidly removed from the circulation would tend to collect together and form the nuclei for clots or become impacted in the small vessels. In the early stages the animal would be able to cope with this, but each relapse would diminish its strength until eventually the danger became insurmountable. Thus the sudden death frequently observed in small animals would be accounted for by cerebral thrombosis; and death in the case of horses by the occurrence of intracardiac clots.

In the lungs parasites were found, though in rather less abundance than in the heart's blood.

In the liver parasites were as numerous as in the heart's blood, occasionally more so.

In the spleen trypanosomes tend to disappear very rapidly after death.

In the kidney parasites were found, although not usually in abundance they were as a rule well defined.

In the glands parasites were invariably absent or scanty.

In brain squashes they were usually scanty although on one occasion a cerebral capillary was found to be occluded by them.

In the bone-marrow trypanosomes could be found but there was no evidence to incriminate this as a site for their evolution.

Animals have been killed when the trypanosomes were just beginning to appear in the peripheral blood and when they were just about to disappear, careful examination has shown that no organ was at either of these times a special seat of the parasites.

All the evidence goes to show that the trypanosomes pass their entire life cycle in a fluid medium, the blood. Each cycle or proliferation is attended by an enormous destruction. A few will remain to produce the succeeding cycle and so on until as suggested some form of fatal clotting or thrombosis occurs or the animal sinks from inanition.

Bitting Flies.

Of the flies which belong to the family Tabanidae, representatives of the genera *Tabanus*, *Chrysops*, and *Hæmatopota* have so far been obtained. Of the flies which belong to the genus *Stomoxys* several species have been found. Only one species of the genus *Hippobosca* has been met with.

Flies of the genera *Tabanus* and *Stomoxys* are plentiful; flies of the genera *Chrysops* and *Hæmatopota* are much less frequently met with, and flies of the genus *Hippobosca* are rare.

Of the flies which belong to the genus *Tabanus* three species are common; the most abundant is a fly with a reddish coloured body, it has been identified as *Tabanus fumifer*, Walker, and is frequently found in numbers in and near jungle, also where cases of Surra have been discovered.

The two other species have greyish striped bodies and are somewhat smaller than those of the preceding species, it has now been determined that they belong to either the species *Tabanus partitus* or *Tabanus vagus* Walker. Flies of these species are found in much less numbers than are the red species. A small species of *Tabanus* (*Tabanus minimus*) has also been found.

Flies of the genus *Stomoxys* can be obtained almost anywhere and at any time, there are numerous species of these flies, but two species are common, namely, *Stomoxys nigra* and *Stomoxys calcitrans*.

The usual mode of obtaining a number of biting flies was to turn a bullock loose near the edge of the jungle between 5 and 6 p.m. Flies would then swarm out and could easily be caught with a net. A certain number would feed or obtain a partial feed but the majority were caught unfed.

Attempts were made to breed these insects, flies of the genus *Tabanus* have on several occasions laid their eggs in ordinary cages and in cages containing growing grass and padi, the larvae have hatched out but these have died. As all experiments to raise presumably infection free flies failed, the experiment had to be conducted with biting flies caught in various places.

In view of the fact that the flies might harbour flagellates or be infected with trypanosomes dissections were made of a large number, mainly *Tabanus fumifer* and film preparations made of their gastrointestinal contents; these were then stained and examined; when the fly had not fed for some time previously such an examination did not present difficulties, but when the insect was distended with blood an accurate search was extremely difficult.

A considerable number of the flies were found to harbour oval shaped parasites, from 6m. to 8m. by 5m. to 6m. whose protoplasm stained a light blue, there was present a nucleus, and a rounded blepharoplast. These parasites were frequently present in considerable clumps or zooglea-like masses, but they have been observed isolated. Examinations of flies infected with these parasites have shown that some hours after they have fed on normal blood a flagellum originates from the centrosome of the oval body which becomes more elongated, until later a distinct flagellated parasite (*Herpetomonas*) has developed. Examinations of the contents of flies fed on infected blood have shown that trypanosomes did not undergo multiplication but gradually underwent involution. At times circular forms were seen but these were invariably smaller than the oval parasites just described and their protoplasm stained badly, the impression formed was that they were but stages in the involution of the trypanosomes and had no connection with the oval parasites, a view which was strengthened by the inoculation experiments made with the contents of biting flies.

Observations under natural conditions have shown that a fly is seldom allowed to complete its meal undisturbed on one animal, because the latter makes every efforts to remove it, the fly however is most persistent in its attentions and may eventually obtain its fill on the one animal, but where

there are several or a herd, the necessary food may be obtained from two or more animals; should the first portion of the meal be obtained from an animal with trypanosomes in its peripheral blood and the second portion from a healthy animal, infection is readily possible, the fly in this way acting in a manner comparable to an infected hypodermic needle.

Experimentally the chief difficulty has been to get the flies to bite; they have been tried at various times in the day and in rainy, dry, as well as dull weather; as many as forty or fifty would be tried at one time and probably none would bite on other occasions several would feed. The usual method was to remove the fly from the cage in a beaker which was then inverted over the skin of the animal. Generally speaking if the fly were to bite it would insert its proboscis almost at once, should the part fixed on be unsuitable it would transfer its attentions to another place, but once having obtained the requisite conditions it might continue its meal for 10 to 15 minutes or even somewhat longer. A fly with its proboscis fixed in the skin and feeding was not readily detached provided that the animal kept quiet; forcible removal could be effected without apparent injury to the insect or on the application of a trace of benzene the fly would at once release its hold. It was thus possible to conduct experiments with flies either partially or completely fed. The former were undoubtedly the more useful for experiments on direct transmission because of their desire to complete their meal.

Mechanical Transmission by means of Biting Flies.

I. GREY SPECIES OF *TABANUS* (*Tabanus partitus*, AND *T. Vagus*).

- (a). A fly of one of these species was allowed to feed on a naturally infected bullock (Exp. 51) which was in an advanced stage of the disease and in whose blood the parasites were scanty. The insect was immediately transferred to a rabbit (Exp. 350) on which feeding was repeated, but no blood flowed from the several punctures made. Daily examinations of the rabbit's blood were thereafter made and trypanosomes found on the eighth day; the rabbit died on the 57th day.
- (b). Two flies of these species were fed on a naturally infected bullock (Exp. 51) one day previous to the death of that animal; parasites were then plentiful in the peripheral blood. On transference to a horse (Exp. 280) feeding was repeated after the insects had rested for about five minutes. Trypanosomes were found on the 9th day in the blood of the horse which died after 77 days having shown the symptoms characteristic of Surra.
- (c). A fly of one of these species was fed on a naturally infected bullock (Exp. 51) eight days previous to the death of that animal; parasites could not be found by microscopic examination. The insect was then allowed to feed on a rabbit (Exp. 351) whose blood was examined thereafter for 54 days with negative results.

II. RED SPECIES OF TABANUS (*Tabanus fumifer*).

- (a). A fly of this species was fed on a naturally infected bullock (Exp. 51) twelve days previous to the death of that animal and in whose blood parasites were then plentiful. On transferring the fly to a rabbit (Exp. 349) feeding was repeated. Examination of the blood for 54 days was made with negative results.
- (b). Three flies of this species were fed on a horse (Exp. 280) which was in the fifth day of the disease and in whose blood parasites could not then be found. On transference to a rabbit (Exp. 356) feeding was repeated. Examination of the blood was made for 31 days with negative results.
- (c). A fly of this species was fed on a horse (Exp. 280) when in the 10th day of the disease; parasites were then plentiful. The fly was transferred to a rabbit (Exp. 357) on whom the fly effected several punctures. Eight days later trypanosomes were found in the blood of the rabbit which died in 57 days.
- (d). A fly of this species was fed on a horse (Exp. 280) four days previous to the death of that animal and in whose blood parasites were then plentiful. Transferred to a bullock (Exp. 373) the feeding was repeated; the blood thereafter was examined for 25 days with negative results. Later the bullock developed trypanosomiasis as the result of subcutaneous injection with blood from this horse.
- (e) A fly of this species was fed on a naturally infected bullock (Exp. 376) ten days previous to the death of that animal and in whose blood parasites could not then be found. The fly was thereafter fed on a rabbit (Exp. 381) whose blood was examined on 33 successive days with negative results.

III. SMALL SPECIES OF TABANUS. (*Tabanus minimus*)

- (a). A fly of this species was fed on a horse (Exp. 384) which was then in the third day of the disease and in whose blood the parasites were scanty. The fly was thereafter fed on a dog (Exp. 385). Two days later this experiment was repeated. Eight days later a fly of this species was fed on a bullock (Exp. 386) which was in the fourth day of the disease and in whose blood parasites were plentiful. The fly was next fed on the dog. Seven days later trypanosomes were found in the blood of the dog which died on the twelfth day.

IV. FLIES OF THE GENUS STOMOXYS.

- (a). A fly of this genus was allowed to feed on a horse (Exp. 280) which was in the twenty-fifth day of the disease and in whose blood trypanosomes were plentiful, it was then fed on a rabbit (Exp. 361) whose blood was examined for 25 days with negative results.

- (b). Two flies of this genus were partially fed on a horse (Exp. 280) which was in the thirtieth day of the disease and in whose blood trypanosomes were plentiful; they were then allowed to complete their feeding on a rabbit (Exp. 364). 11 days later and when the parasites were swarming in the blood of the horse, this experiment was repeated with another two flies of this genus. The blood of the rabbit was examined on 50 consecutive days with negative results.
- (c). Eleven flies of this genus were fed on a horse (Exp. 280) which was in the fifty-seventh day of the disease and in whose blood parasites were plentiful; the flies were then fed on another horse (Exp. 366). Examination of this horse's blood was thereafter made for 40 days with negative results.
- (d). Three flies of this genus were fed on a horse (Exp. 280) seven days previous to the death of that animal and in whose blood trypanosomes were scanty. The flies were then fed on a rabbit (Exp. 372). The blood of the rabbit was thereafter examined for 25 days with negative results.
- (e). Eight flies of this genus were fed on a dog (Exp. 436) which was then in the third day of the disease and in whose blood parasites were few. The flies were then fed on a rabbit (Exp. 440) on the same day four flies of this genus were fed on a dog (Exp. 431) which was in the thirtieth day of the disease and in whose blood parasites were plentiful. The flies were next fed on the rabbit whose blood was examined for 35 days with negative results.

V. FLIES OF THE GENUS *HÆMATOPOTA*.

- (a). A fly of this genus was fed on a horse (Exp. 280) which was in the twenty-fifth day of the disease and in whose blood parasites were plentiful; the fly was thereafter fed on a rabbit (Exp. 361) whose blood was examined for twenty-five days with negative results.
- (b). A fly of this genus fed on a horse (Exp. 384) whose death took place on the following day and in whose blood parasites were plentiful. The fly was then fed on a guinea-pig (Exp. 413) whose blood was examined for thirty days with negative results.

Attempt to transmit trypanosomiasis by means of biting flies which had fed on an affected animal 48 to 72 hours previously.

It was found extremely difficult to get flies to bite when 48 to 72 hours had elapsed since their previous feed. Nearly 400 flies were tried, only 38 of them fed on the infected horse; the experiment extended over six weeks.

Five flies of the grey species of *Tabanus* and four flies of the red species fed on a horse (Exp. 280) in whose blood at the time parasites were plentiful. Forty-eight hours later these flies fed on a Javanese pony (Exp. 347)

Two flies of the grey species of *Tabanus* and two flies of the red species fed on a horse (Exp. 280) in whose blood at the time parasites were plentiful. Seventy-two hours later these flies fed on the Javanese pony.

As by neither of these experiments had the pony become infected, flies of the genus *Tabanus* were fed on the horse (Exp. 280) and after 48 hours let loose in the stall occupied by the Java pony. In all 21 flies of the grey species of *Tabanus* and 4 flies of the red species were so dealt with. It is impossible to say if any of these flies fed on the pony. After five or six days most of the flies were dead. The blood of the Java pony was regularly examined for over two months but on no occasion were trypanosomes found.

Inoculation Experiments.

Trypanosomes when ingested by a biting fly appear for the most part at least, as already stated to undergo involution and death, the rounded forms referred to might possibly belong to another stage in the life history of the parasite and it was possible that part of the cycle of the trypanosomes was carried out in the flies. According to this view trypanosomes so long as they were present as such in the gastro-intestinal contents would be capable of being inoculated and exciting the disease in a susceptible animal, but afterwards they would be unable to do so until they had undergone a process of development probably sexual.

If it was difficult to get flies to bite when freshly caught it was found still more difficult to get them to feed again after an interval of 24 hours or more, and as it was impracticable to make a complete examination of the contents of such insects it was decided to keep flies which had fed on infected blood for periods ranging from 24 hours to 14 days; when the required interval had elapsed the fly was killed with chloroform, the wings and legs removed; the body was then opened, the entire contents were scraped out, cut up in normal saline solution by means of a fine scissors and the resulting emulsion was injected into a guinea-pig. In this way it was found possible to include practically everything save the chitinous covering, and the interval between the death of the fly and the inoculation of its contents into a susceptible animal could be reduced to a few minutes.

Difficulty was experienced in keeping the infected flies alive in captivity and although various plans have been tried only partial success has attended those efforts. It has not been possible to keep infected flies alive for 14 days, on one occasion an infected fly did live for 13 days but on the following morning it was found dead in the cage.

Inoculation after 24 hours.

I. RED SPECIES OF *TABANUS*.—(*Tabanus fumifer*.)

- (a) A fly of this species was used which had fed on a horse (Exp. 280) one day previous to the death of that animal and in whose blood trypanosomes were then scanty. A rabbit (Exp. 375) was used for the inoculation but trypanosomes were not found on daily examination up to the 17th day when death occurred apparently from septicaemia.
- (b) A fly of this species was used which had fed on a naturally infected bullock (Exp. 51) seven days previous to the death of that animal and in whose blood by microscopic exami-

nation trypanosomes could not then be found. A guinea-pig (Exp. 382) was used and the inoculation was made subcutaneously. On the 15th day parasites were found and death took place on the 90th day.

- (c) A fly of this species fed on a horse (Exp. 384) which was in the 12th day of the disease and in whose blood there were then but few parasites. A guinea-pig (Exp. 389) was used and an intraperitoneal inoculation was made. On the 12th day trypanosomes were found and death occurred on the 68th day.
- (d) A fly of this species fed on a horse (Exp. 384) which was in the 4th day of the disease and in whose blood parasites were then few. Intraperitoneal inoculation was made on a guinea-pig (Exp. 393) which died after 16 days without trypanosomes having on any day been found in the blood.
- (e) A fly of this species fed on a naturally infected pony (Exp. 432) which was then in the 20th day of the disease and in whose blood trypanosomes were plentiful. Intraperitoneal inoculation was made on a guinea-pig (Exp. 438) which did not develop trypanosomiasis.

II. GREY SPECIES OF TABANUS. (*Tabanus partitus* AND *T. vagus*.)

- (a). A fly of one of these species fed on a horse (Exp. 384) which was then in the 45th day of the disease and in whose blood the parasites were swarming. Intraperitoneal inoculation was made on the guinea-pig (Exp. 408) which has not developed trypanosomiasis.
- (b). A fly of one of these species was fed on a naturally infected pony (Exp. 432) which was then in the 25th day of the disease and in whose blood parasites were then plentiful. Intraperitoneal inoculation was made on a guinea-pig (Exp. 439) which has not developed trypanosomiasis.

Inoculation after 48 hours.

I. RED SPECIES OF TABANUS. (*Tabanus fumifer*.)

A fly of this species was fed on a horse (Exp. 384) which was then in the 13th day of the disease and in whose blood parasites were few. Intraperitoneal inoculation was made on a guinea-pig (Exp. 390) which has not developed trypanosomiasis.

II. GREY SPECIES OF TABANUS. (*Tabanus partitus* AND *T. vagus*.)

A fly of one of these species was fed on a horse (Exp. 384) which was then in the 43rd day of the disease and in whose blood trypanosomes were swarming. Intraperitoneal inoculation was made on a guinea-pig which did not develop trypanosomiasis.

Inoculation after 72 hours.

I. RED SPECIES OF TABANUS. (*Tabanus fumifer*.)

A fly of this species was fed on a horse (Exp. 384) which was then in the 15th day of the disease and in whose blood trypanosomes were plentiful. Intraperitoneal inoculation was made on a guinea-pig (Exp. 394) in whom 62 subsequent daily examinations have failed to find trypanosomes.

II. GREY SPECIES OF TABANUS. (*Tabanus partitus* AND *T. vagus*.)

A fly of one of these species was fed on a naturally infected pony (Exp. 432) which was then in about the 20th day of the disease and in whose blood trypanosomes were plentiful. Intraperitoneal inoculation was made on a guinea-pig (Exp. 437) which has not developed trypanosomiasis.

Inoculation after 96 hours.

I. RED SPECIES OF TABANUS. (*Tabanus fumifer*.)

A fly of this species was fed on a horse (Exp. 384) which was in the 16th day of the disease and in whose blood trypanosomes were plentiful. Intraperitoneal inoculation was made on a guinea-pig (Exp. 396) in whom 60 subsequent daily examinations have failed to find trypanosomes.

Inoculation after 120 hours.

I. RED SPECIES OF TABANUS. (*Tabanus fumifer*.)

A fly of this species was fed on a horse (Exp. 384) which was then in the 20th day of the disease and in whose blood trypanosomes were plentiful. Intraperitoneal inoculation was made on a guinea-pig (Exp. 401) in whom 69 subsequent daily examinations have failed to find trypanosomes.

Inoculation after 7 days.

I. GREY SPECIES OF TABANUS. (*Tabanus partitus* AND *T. vagus*.)

A fly of one of these species was fed on a naturally infected pony (Exp. 432) which was then in about the 25th day of the disease and in whose blood trypanosomes were plentiful. Intraperitoneal inoculation was made on a guinea-pig which has not developed trypanosomiasis.

II. RED SPECIES OF TABANUS. (*Tabanus fumifer*.)

A fly of this species was fed on a horse (Exp. 458) in whose blood trypanosomes were then scanty. Intraperitoneal inoculation was made on a guinea-pig (Exp. 462) which did not develop trypanosomiasis.

Inoculation after 10 days.

I. RED SPECIES OF TABANUS. (*Tabanus fumifer*.)

A fly of this species was fed on a dog (Exp. 431) which was in the 31st day of the disease and in whose blood trypanosomes were then plentiful. Intraperitoneal inoculation was made on a guinea-pig which has not developed trypanosomiasis.

Treatment.

The encouraging results, which have been recorded by various investigators, from the combined use of Atoxyl and Mercuric Chloride, induced us to test the value of this treatment.

Atoxyl was employed in the form of a 10 % solution which was freshly prepared for each injection, and given either subcutaneously or intravenously.

The Mercuric Chloride was dissolved in salt solution and was given as an intramuscular injection.

EXPERIMENT 432.

A Javanese pony, 5 years old, and about 12 hands high, was found naturally infected with the disease. It was stated to have been ill for five or six days previous to its coming under observation. The animal appeared very dull, there was œdema of the legs, sheath, and dependent part of the abdomen, trypanosomes were present in fair abundance, in addition microfilariæ were noted. It was removed to the Institute for observation. The œdema became more and more marked, loss of condition pronounced. and there was weakness of the hindquarters. Twelve days after the animal was found, treatment commenced and was carried out as shown in the table, which also shows the results of the microscopical examination of the blood for trypanosomes.

The Atoxyl was administered subcutaneously.

| Date. | Treatment. | Temperature. | | Microscopical
estimation of the
parasites in the
peripheral
blood. |
|------------|----------------------------------|--------------|-------|--|
| | | M. | E. | |
| Sept. 26th | 0.5 grms. Atoxyl ... | 103.4 | 103.8 | Plentiful |
| " 27th | 0.5 " " ... | 103 | 102 | Absent |
| " 28th | No treatment ... | 102 | 103 | " |
| " 29th | 1 grm. Atoxyl ... | 101.4 | 101.6 | " |
| " 30th | 0.05 grms. Mercuric Chloride ... | 100 | 100.4 | " |
| Oct. 1st | No treatment ... | 100.4 | 101.6 | " |
| " 2nd | 1 grm. Atoxyl ... | 101 | 100 | " |
| " 3rd | 0.08 " Mercuric Chloride ... | 101.2 | 102 | " |
| " 4th | 1 " Atoxyl ... | 100 | 102 | " |
| " 5th | 0.08 " Mercuric Chloride ... | 100.2 | 101 | " |
| " 6th | 1 " Atoxyl ... | 100.4 | 101 | " |
| " 7th | 0.08 " Mercuric Chloride ... | 102 | 100 | " |
| " 8th | 2 " Atoxyl ... | 101.4 | 100.6 | Few |
| " 9th | 0.1 " Mercuric Chloride ... | 102 | 105 | Scanty |
| " 10th | 2 grms. Atoxyl ... | 100.4 | 102 | Absent |
| " 11th | 0.1 grm. Mercuric Chloride ... | 100 | 100 | " |
| " 12th | No treatment ... | 100.2 | 100 | " |
| " 13th | 0.08 grm. Mercuric Chloride ... | 100.2 | 100.8 | " |
| " 14th | 1.5 " Atoxyl ... | 100.4 | 100.2 | " |
| " 15th | 0.08 " Mercuric Chloride ... | 100.4 | 100 | " |
| " 16th | No treatment ... | 100.8 | 101 | " |
| " 17th | " " ... | 100.8 | 100.2 | " |
| " 18th | " " ... | 101.2 | 100.8 | " |
| " 19th | 1.5 grm. Atoxyl ... | 100.8 | 100.2 | " |
| " 20th | No treatment ... | 102.4 | 100.8 | " |
| " 21st | 0.08 grm. Mercuric Chloride ... | 100.8 | 101 | " |
| " 22nd | 0.08 " " ... | 100.2 | 101 | " |
| " 23rd | 1.5 " Atoxyl ... | 100.8 | 101 | " |
| " 24th | 2 grms. Atoxyl ... | 106.4 | 105.8 | Few |
| " 25th | 0.1 grm. Mercuric Chloride ... | 100.6 | 103.8 | Absent |
| " 26th | 2 grms. Atoxyl ... | 101.4 | 101 | " |
| " 27th | 0.15 grm. Mercuric Chloride ... | 101 | 101 | " |

| Date. | | Treatment. | | Temperature. | | Microscopical
estimation of the
parasites in the
peripheral
blood. |
|-----------|-----------------------|------------------------|-----|--------------|-------|--|
| | | | | M. | E. | |
| Oct. 28th | 3 | grms. Atoxyl | ... | 101 | 101·4 | Absent. |
| " 29th | 0·15 | grm. Mercuric Chloride | ... | 100·2 | 100 | " |
| " 30th | 2 | grms. Atoxyl | ... | 100·4 | 100 | " |
| " 31st | 0·15 | grm. Mercuric Chloride | ... | 99·8 | 100 | " |
| Nov. 1st | 3 | grms. Atoxyl | ... | 100·8 | 100 | " |
| " 2nd | 0·15 | grm. Mercuric Chloride | ... | 100·4 | 100 | " |
| " 3rd | 3 | grms. Atoxyl. | ... | 100·2 | 99·8 | " |
| " 4th | 0·2 | " Mercuric Chloride | ... | 100 | 100·4 | " |
| " 5th | 3 | " Atoxyl | ... | 100·2 | 99·6 | " |
| " 6th | 3 | " Atoxyl | ... | 100·4 | 100·2 | " |
| " 7th | 0·15 | grm. Mercuric Chloride | .. | 100·2 | 100 | " |
| " 8th | 2 | grms. Atoxyl | ... | 100·4 | 100·8 | " |
| " 9th | 0·1 | grm. Mercuric Chloride | ... | 100·4 | 100 | " |
| " 10th | 3 | grms. Atoxyl | ... | 100·2 | 100·6 | " |
| " 11th | 0·15 | grm. Mercuric Chloride | ... | 100·2 | 100 | " |
| " 12th | 2 | grms. Atoxyl | ... | 100·6 | 101 | " |
| " 13th | 0·1 | grm. Mercuric Chloride | ... | 100·4 | 100 | " |
| " 14th | No treatment | .. | ... | 99·2 | 99·8 | " |
| " 15th | No treatment | ... | ... | 100·6 | 99 | " |
| " 16th | Died during the night | ... | ... | | | |

The oedema gradually lessened in amount, and eventually disappeared about 14 days after the commencement of treatment, the pony was then much brighter and there was no longer evidence of weakness, the abdomen, however, became very much distended. The appetite was good and the bowels acted normally throughout. Emaciation continued to increase in spite of a liberal dietary. During the last week large petechiæ were observed on the conjunctival mucosa.

Two days previous to death, the pony was found lying down, it was able to get up but appeared dull and disinclined for exertion, weakness of the hindquarters was once more evident.

The next day it was unable to rise, and died during the night. Post-mortem; "goose-fat" clots were found in the cavities of the heart, otherwise there was nothing of note. Smears were made from the heart's blood, the lungs, spleen, liver, kidneys, and bone marrow, they were stained and examined, but no trypanosomes or anything resembling them could be found.

A rat, which was inoculated with 2 c.c. of blood drawn from the jugular vein of the pony the day before death, did not become infected.

EXPERIMENT 450.

An Australian mare, aged, and in condition was inoculated with 0·5 c.c. of infected blood; trypanosomes appeared on the fifth day and 10 days later, when the clinical signs of the disease were marked, treatment was commenced. Atoxyl was administered subcutaneously in much larger

doses than in the preceding case, and an injection of Mercuric Chloride was only once given:—

| Date. | | Treatment. | | Temperature. | | Microscopical
estimation of the
parasites in the
peripheral
blood. |
|----------|----------|---------------------------------|-----|--------------|-------|--|
| | | | | M. | E. | |
| October | 28th ... | 4 grms Atoxyl | ... | 103 | 104 | Plentiful. |
| " | 29th ... | 4 " " | ... | 100 | 101·8 | Absent |
| " | 30th ... | No treatment | ... | 101 | 101·4 | " |
| " | 31st ... | 4 grms. Atoxyl | ... | 101 | 101·6 | " |
| November | 1st ... | No treatment | ... | 101·4 | 100·6 | " |
| " | 2nd ... | 5 grms Atoxyl | ... | 100·6 | 100·8 | " |
| " | 3rd ... | No treatment | ... | 101 | 101·6 | " |
| " | 4th ... | 5 grms. Atoxyl | ... | 100·8 | 101·6 | " |
| " | 5th ... | 5 " " | ... | 101 | 100·6 | " |
| " | 6th ... | 5 " " | ... | 101·2 | 100 | " |
| " | 7th ... | 5 " " | ... | 101·2 | 101·6 | " |
| " | 8th ... | 0·25 grms. Mercuric
Chloride | ... | 100·8 | 101·4 | " |
| " | 9th ... | 5 grms. Atoxyl | ... | 100·6 | 99·8 | " |
| " | 10th ... | 5 " " | ... | 100·2 | 100·8 | " |
| " | 11th ... | No treatment | ... | 100·6 | 101 | " |
| " | 12th ... | " " | ... | 100·2 | 102·6 | " |
| " | 13th ... | " " | ... | 101 | 102 | " |
| " | 14th ... | " " | ... | 102·4 | 102·2 | " |
| " | 15th ... | " " | ... | 103·6 | 103 | " |
| " | 16th ... | " " | ... | 105 | 104·8 | " |
| " | 17th ... | " " | ... | 105 | 105·8 | " |
| " | 18th ... | " " | ... | 104·4 | 104 | " |
| " | 19th ... | " " | ... | 103·8 | 104·4 | " |
| " | 20th ... | " " | ... | 103·2 | 102 | " |
| " | 21st ... | Mare died 10 a.m. | | | | |

The clinical signs gradually became less pronounced and the animal bore the treatment well until the 11th November, when she showed symptoms of colic; later she appeared very distressed at intervals and suddenly collapsed and fell down as if shot, she would remain thus for a few minutes, and then rise apparently recovered; these attacks continued during that day. The next morning she was found to be very badly bruised, there were large wounds on the knees and fetlocks, all four legs were much swollen; she could stand but if made to move at once collapsed. Treatment was discontinued, the mare began to improve and was eating well, but on the 21st November death suddenly occurred.

A post-mortem examination was made immediately after death but nothing of note was observed.

A dog, which was inoculated with 10 c.c. of blood drawn from the jugular vein shortly before death, did not develop trypanosomiasis.

EXPERIMENT 458.

An Australian gelding, aged and in fair condition, was inoculated with 3 c.c. of blood from a naturally infected bullock. On the fourth day

trypanosomes were found in the peripheral blood, fourteen days later treatment was commenced, the clinical signs were then marked.

Atoxyl was administered intravenously in this case, and on three occasions an intravenous injection of Tartarated Antimony was given.

| Date. | Treatment. | | Temperature. | | Microscopical estimation of the parasites in the peripheral blood. |
|------------------|------------------------------|-----|--------------|-------|--|
| | | | M. | E. | |
| December 8th ... | 5 grms. Atoxyl | ... | 104·6 | 105 | Plentiful |
| " 9th ... | 5 " " | ... | 99·6 | 100·6 | Absent |
| " 10th ... | No treatment | ... | 99·8 | 100 | " |
| " 11th ... | 0·25 grms. Mercuric Chloride | ... | 100·4 | 101 | " |
| " 12th ... | 0·25 " " | ... | 100 | 99·8 | " |
| " 13th ... | 0·25 " " | ... | 100·4 | 101 | " |
| " 14th ... | 5 grms. Atoxyl | ... | 100·6 | 100 | " |
| " 15th ... | 0·25 grms. Mercuric Chloride | ... | 100·8 | 100·6 | " |
| " 16th ... | No treatment | ... | 100·6 | 101 | " |
| " 17th ... | 0·25 grms. Mercuric Chloride | ... | 100·8 | 100·4 | " |
| " 18th ... | No treatment | ... | 101 | 101·4 | " |
| " 19th ... | 0·25 grms. Mercuric Chloride | ... | 100·8 | 100 | " |
| " 20th ... | No treatment | ... | 101 | 100·6 | " |
| " 21st ... | 0·25 grms. Mercuric Chloride | ... | 101·2 | 101·6 | " |
| " 22nd ... | No treatment | ... | 102 | 102·2 | " |
| " 23rd ... | 2 grms. Tartarated Antimony | ... | 101·8 | 102 | Scanty |
| " 24th ... | No treatment | ... | 101·2 | 101 | Absent |
| " 25th ... | " " | ... | 100·6 | 100·4 | " |
| " 26th ... | 2·5 grms. Atoxyl | ... | 101·2 | 100·6 | " |
| " 27th ... | No treatment | ... | 101 | 100·8 | " |
| " 28th ... | 2·5 grms. Atoxyl | ... | 100·6 | 100·8 | " |
| " 29th ... | No treatment | ... | 100·4 | 101 | " |
| " 30th ... | " " | ... | 100·6 | 100 | " |
| " 31st ... | 1 grm. Tartarated Antimony | ... | 100·4 | 101 | " |
| January 1st ... | No treatment | ... | 100·4 | 100 | " |
| " 2nd ... | " " | ... | 99·8 | 100 | " |
| " 3rd ... | 2·5 grms. Atoxyl | ... | 100·2 | 100·8 | " |
| " 4th ... | No treatment | ... | 100·2 | 100 | " |
| " 5th ... | " " | ... | 99·6 | 99·8 | " |
| " 6th ... | " " | ... | 100·6 | 101 | " |
| " 7th ... | " " | ... | 100 | 100·6 | " |
| " 8th ... | 1 grm. Tartarated Antimony | ... | 100·4 | 100·8 | " |
| " 9th ... | Treatment stopped | ... | | | |

The first injections of Atoxyl induced slight symptoms of colic as also did the first injection of Tartarated Antimony but the subsequent injections produced no apparent disturbance.

About the beginning of January the general health of the horse had improved and there were no clinical signs of the disease; on the 9th January treatment was suspended, trypanosomes had then been absent for 17 days. On the 24th December the temperature was normal and remained so until the 20th January, when a febrile attack occurred which lasted for four days, trypanosomes were not found. From the 24th to the 27th January the temperature was normal, thereafter the temperature continued elevated and irregular. On the 3rd February trypanosomes returned. On the 12th February the animal died of Surra.

Treatment by means of Atoxyl and Mercuric Chloride was also tried on several dogs affected with trypanosomiasis but with unfavourable results.

Conclusions.

The trypanosomes which have been the subject of this investigation belong to the species *Trypanosoma Evansi*.

The trypanosomes which have been met with in horses, cattle, and dogs are indistinguishable morphologically and in their pathological effects.

Considerable variations have been noted in the virulence of the several strains of trypanosomes dealt with.

The clinical diagnosis of the disease in horses is comparatively easy.

The disease in cattle is to be suspected when there is marked emaciation which cannot otherwise be accounted for; as so great difficulty is frequently encountered in the detection microscopically of trypanosomes in cattle, inoculation of their blood into laboratory-animals will as a rule be required.

Mechanical transmission of the disease has been effected by four species of flies of the genus *Tabanus*, but not by flies of the genus *Stomoxys*.

Surra has only been met with in horses whose stables were in the vicinity of jungle, and consequently where flies of the species *Tabanus fumifer* abound.

In cattle the history usually points to the infection having been acquired in the outlying districts.

In towns there are a certain proportion of infected animals and numbers of susceptible ones but flies of the genus *Tabanus* are not common.

Experimentally it has been found that the incubation period is intimately connected with the number of parasites inoculated and the rate of proliferation of the trypanosomes in the animal; *ceteris paribus*, the fewer the trypanosomes inoculated the longer will be the interval before their presence is discovered in the peripheral blood and *vice versa*.

Experiments conducted with a view to determine whether or not part of the life cycle of the trypanosomes was carried out in biting flies, were inconclusive, but so far as they have gone it has been shown that trypanosomes, as such, are not to be found in the gastro-intestinal contents of infected flies after 24 hours.

Treatment by means of Atoxyl, Mercuric Chloride, and Tartarated Antimony, was not encouraging. The parasites could be caused to disappear from the peripheral circulation and the health of the animals much improved but these results were only temporary.

Surra is endemic in the Federated Malay States; in addition, animals harbouring the parasites may be and have been imported from India, Siam, and the Siamese States, as well as Java and Sumatra.

No practical measures are available whereby the entry of apparently healthy cattle harbouring the parasites might be prevented.

In the present state of our knowledge it is scarcely possible to hope for eradication of the disease, it can however be kept in check by the destruction of all animals found to be affected.

Emaciated cattle, whose condition cannot be accounted for, should be destroyed.

The herding of cattle in large numbers, especially near jungle is to be avoided.

The detention of horses and cattle at the port or place of entry into these States, until such time as they have been examined is advisable.

Quarantine, as usually understood, is unlikely to be of assistance in the detection of the disease.

Distribution of certain species of Biting Flies in the Federated Malay States.

(TABANIDÆ AND STOMOXYS)

By

H. C. PRATT, GOVERNMENT ENTOMOLOGIST.

In connection with the work on Surra a large number of flies of the Family Tabanidæ and of the genus-Stomoxys have been obtained in various parts of the Federated Malay States.

At present a full account of the distribution of these flies is not possible as the length of time spent in the places visited has been insufficient. Nevertheless the collections formed indicate that not only are certain species or varieties confined to comparatively small areas, but the limitation of some species in certain places is very marked. This localization of species and varieties as well as their prevalence or rarity appears to be affected by the nature of the country, whether:—Jungle, Bush, Bluker* or Town. Nearly thirty species of Tabanidæ have been collected, and these are referable to three genera:—*Tabanus*, *Chrysops*, and *Haematopota*. The majority of species are as yet undescribed; those identified are as follows:—

Tabanus fumifer Wlk: *T. opatus* Wlk: *T. minimus* V. D. Wulp. *T. partitus* Wlk: *T. vagus* Wlk: *Chrysops fixissimus* Wlk: *C. flavocinctus* Ricardo. *C. dispar* Fabricius. *Haematopota atomaria* Wlk; *H. rubida* Ricardo and *H. unizonata* Ricardo.

Of the genus *Tabanus* the only species which are peculiar so far as their distribution is concerned are *T. fumifer* and *T. partitus*. The former of these two species is undoubtedly the predominant and most widely distributed Tabanid in these States; it presents numerous varieties, and some of these appear to be confined to small areas.

A typical form of *T. fumifer* has a series of well developed inverted V shaped markings placed on the dorsum of the abdomen at the apical portion of each segment; the colour of the abdomen in such specimens is chocolate brown. This form has up to the present only been obtained in the towns and their outskirts; it does not occur in abundance.

The other forms of *T. fumifer* differ chiefly in the markings of the abdomen; the varieties which have these markings imperfectly represented are to be found in comparatively open places, and they frequent the edges of jungle, occurring in large numbers on roads bounded on either side by dense forest. The following record of the number of flies of this species captured in two evenings between the hours of 5 and 6 p.m. will give some idea of their prevalence in such places.

* A term applied to a second growth which has sprung up on land previously cleared of the virgin jungle.

| | No. of
specimens |
|--|---------------------|
| Inverted V shaped markings absent | 206 |
| " " " " present on one segment ... | 1 |
| " " " " " " hinder segments ... | 1 |
| " " " " just visible on all segments ... | 185 |
| " " " " rather more distinct ... | 9 |
| | <hr/> 402 |

The place where these flies were obtained cannot be described as dense forest country; practically the only forest left is that which has been reserved by the Forest Department, the rest has been felled and is now planted with coffee and rubber.

Generally speaking it would appear that *T. fumifer* prefers comparatively open country interspersed with tracts of dense forest to the jungle of the Malay Peninsula. Its absence in and around large towns, and its prevalence in the smaller villages and along the edges of the jungle bordering the roads is apparently to be accounted for by the fact that in the two latter places the breeding grounds have not been interfered with to any great extent, and also because of the presence of bullocks, the blood of which, although not a necessary means of sustenance undoubtedly affords a suitable food for the females of Tabanidae. The combination of these favourable factors has a decided influence on the prevalence of *T. fumifer*.

The following table which is a record of the species of Tabanidae obtained in eight days during five different months shows the predominance of certain species over others. The specimens were not all obtained in the same place.

| | | | |
|------------------------|-----|-----|------|
| <i>T. fumifer</i> | ... | ... | 642 |
| <i>T. opatus</i> | ... | ... | none |
| <i>T. minimus</i> | ... | ... | 140 |
| <i>T. partitus</i> | ... | ... | none |
| <i>T. vagus</i> | ... | ... | 20 |
| <i>C. fixissimus</i> | ... | ... | none |
| <i>C. flavocinctus</i> | ... | ... | " |
| <i>C. dispar</i> | ... | ... | " |
| <i>H. atomaria</i> | ... | ... | " |
| <i>H. rubida</i> | ... | ... | 2 |
| <i>H. uizonata</i> | ... | ... | none |

The typical form of *T. fumifer* which occurs in towns has not yet been observed in the dense forest country where very few *T. fumifer* are to be obtained. It is however not possible to make a definite statement regarding this as very little time has been devoted to the forest country.

Those species of Tabanidae that occur in the large towns do not appear to breed to any extent within a four mile radius of the town. This is undoubtedly due to the operations of man; the jungle has been felled, the land drained, the rivers are frequented by buffaloes and the natives utilize these rivers to wash their cattle, factors which have either abolished, diminished or rendered these places unfavourable to the propagation of Tabanidae. The diminution of these breeding grounds

consequent on the causes just mentioned has reference at the present time to about four places in the Federated Malay States. Bullocks which have their sheds in the larger towns, are not confined to these towns, but are chiefly used for the transport of goods to out-stations, and near the roads leading to, as well as in the vicinity of out-stations there are abundant breeding places favourable to the multiplication of Tabanidae; thus these bullocks are as frequently bitten as are those which have their work confined to the country.

The uniform climate of the Malay Peninsula offers facilities for the breeding of Tabanidae the whole year round, and this undoubtedly occurs with *T. fumifer* which may be obtained in almost equal numbers from January to December.

Several of the other species occur somewhat sporadically as for instance *T. partitus* and all the species of *Chrysops* and *Haematopota*.

The distribution of *Chrysops* and *Haematopota* is fairly general; they have been observed in many parts of the Federated Malay States, but are far from common, and on occasion several months pass without a specimen being obtained, thus indicating a breeding season for these genera. *Haematopota* prefers the "blukerland" to the dense jungle of the Malay Peninsula, a fact which accounts for its prevalence in the more civilized districts. *Chrysops dispar* on the other hand appears to show no preference for any special land, but is found in small numbers throughout the Peninsula, although appearing somewhat spasmodically at different times of the year.

Placed approximately in order of abundance as met with on bullocks the arrangement would be: *Tabanus fumifer*, *T. minimus*, *T. partitus*, *T. vagus* (*T. opatus* is a comparatively rare species) *Chrysops flavocinctus*, *C. dispar*, *C. fixissimus*, *Haematopota rubida*, *H. atomaria*, *H. unizonata*. The Tabanidae here are always more abundant during the cooler hours of the day, that is, just after sunrise and shortly before sundown.

STOMOXYS.

The only point of interest in connection with the distribution of flies of this genus is that their occurrence in considerable numbers is confined to those open places which are frequented by cattle, being especially abundant near the cattle sheds between the hours of 10 a.m. and 4 p.m.

So far they have been met with all over the Federated Malay States, but they do not occur to any extent in jungle, nor at the edges of the jungle bordering roads, etc. Their distribution therefore although general is limited to those places where cattle are either kept or where there is pasture.

The most favourable means for the multiplication of *Stomoxys* is afforded in the mining towns where bullocks are utilized for the transport of goods by road; the business transacted in these places is large and necessarily entails the use of numerous bullocks. Thus especially in mining towns, *Stomoxys* may be seen in great abundance and is most active during the hottest hours of the day.

A fairly large collection of flies belonging to this genus has been obtained, consisting of about six species only two of which have as yet been identified, viz., *Stomoxys calcitrans* L. and *S. nigra* Macquart. *S. calcitrans* is probably the commonest species here.

On one occasion a species of *Stomoxys* was found breeding in the diseased roots of padi, beneath a layer of water some 18 inches deep.

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES

No. 10.

AN INQUIRY CONCERNING THE ETIOLOGY
OF

BERI-BERI

BY

HENRY FRASER, M.D. (ABER.);

Director, Institute for Medical Research

AND

A. T. STANTON, M.D., (TOR.)

Bacteriologist, Institute for Medical Research

Printed by Authority of the Resident-General, F.M.S.

Singapore:

KELLY & WALSH, LIMITED, PRINTERS.

SHANGHAI, HONGKONG & YOKOHAMA.

1909.

Price: Three Shillings and Sixpence net.

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES

No. 10.

AN INQUIRY CONCERNING THE ETIOLOGY
OF

BERI-BERI

BY

HENRY FRASER, M.D. (ABER.),

Director, Institute for Medical Research

AND

A. T. STANTON, M.D., (TOR.)

Bacteriologist, Institute for Medical Research

Printed by Authority of the Resident-General, F M S

Singapore:

KELLY & WALSH, LIMITED, PRINTERS,
SHANGHAI, HONGKONG & YOKOHAMA.

1909.

Price: Three Shillings and Sixpence net.

We desire to record the important services rendered by Dr. W. L. BRADDON, lately State Surgeon of Negri Sembilan, in connection with the work on which this report is based. Dr. BRADDON's views respecting the origin of Beri-Beri are well known to students of this disease and it was to test the accuracy of these views that this inquiry was undertaken. It was on Dr. BRADDON's initiative that the approval of Government was obtained. Throughout the course of the investigation Dr. BRADDON was associated with us in the work and generously placed at our disposal his unrivalled knowledge of the clinical and other aspects of the Beri-Beri problem.

H. F.

A. T. S.

25°

La Semantan

test
ible
ted,
nce.
ous

ery
nes
me
eri-

was
ese
and
ave
ms
ges

ere
for
ote.
was
and
nc-
usk
yo,
ed.
the
ong
ose
on

has
eri
the
ng
ale
nd
ied
pre
ler
ice
d,"
the

W.
conr
BRA
to st
view
initii
out
with
unri
Ber:

An Inquiry Concerning the Etiology of Beri-Beri.

It has long been recognized that the incidence of Beri-Beri is greatest among rice-consuming peoples, and whilst from time to time a possible connection between rice eating and this disease has been suggested, hypotheses based on such suggestions have not met with general acceptance. Indeed most authorities have deemed them unworthy of serious consideration.

Certain oriental peoples partake of a diet in which rice bulks very largely; meats and fish are eaten in relatively small amounts and in times of stress these are still further reduced. In view of this fact some observers have suggested that such peoples are underfed and that Beri-Beri is the result of proteid or possibly fat starvation.

Believing that the diet issued to the sailors in the Japanese Navy was deficient in proteid, TAKAKI¹ in 1884 introduced dietetic reforms. These were followed by a remarkable diminution in the number of cases and finally by the disappearance of the disease. Critics of TAKAKI's work have stated that coincident with the alterations in diet, various sanitary reforms were also carried out and that these latter rather than the dietary changes were responsible for the results obtained.

EIJKMAN² as the result of experiments on fowls concluded that there exists commonly in rice a poison which produces polyneuritis, and that for this poison or its effects something contained in the pericarp is an antidote.

An investigation based on the results of EIJKMAN's researches was carried out by VORDERMAN³ in 1895 and 1896 in the prisons of Java and Madoera. In this investigation red and white rices were used. The distinction between red and white rice is that in the case of the former only the husk has been removed whilst in the case of the latter the pericarp, embryo, spermoderm, perisperm and part of the endosperm have been removed. The results indicated that the incidence of Beri-Beri among the prisoners varied directly as the amount of white rice in the diet. Among those on red rice the incidence of Beri-Beri was .01 per mille, among those on a mixture of red rice and white rice 2.4 per mille, and among those on white rice 28 per mille.

BRADDON,⁴ from observations in the Federated Malay States, has drawn attention to the curious discrepancy in the prevalence of Beri-Beri among the immigrant peoples in these States and the Straits Settlements, the vast majority of cases being met with in Chinese and extremely few among Tamils. He believed that the disease was due to the consumption of stale white rice, the staple article of diet among the Chinese immigrants, and that the Tamils remained free from the disease so long as they consumed only rice prepared in the Indian manner, that is by parboiling before husking. A similar immunity from the disease enjoyed by Malays under primitive conditions he believes to be due to the fact that they consume rice prepared from padi newly husked. He has given the names "uncured," "cured" and "fresh" respectively to these forms of rice. This view of the

etiology of Beri-Beri constitutes, if correct, an important advance upon any of the hypotheses hitherto formulated which seek for the origin of the disease in food.

BRADDON has dealt with the whole question in some detail in a recent publication "The Cause and Prevention of Beri-Beri"; among the conclusions arrived at from an analysis of his own observations and the available statistical evidence are the following :—

- (1) "The formation of poison in stale rice is probably due neither to fermentation nor bacteria but to the growth in it of a special fungus."
- (2) "The poison of stale rice has an antecedent in fresh rice. The agent must be therefore some ferment or parasite or epiphyte peculiar to padi."
- (3) "The specific fungus of Beri-Beri is like that of toxic rye and lolium probably a parasite affecting the surface of the seed."
- (4) "The Beri-Beri producing fungus of rice is probably a surface parasite or epiphyte affecting the seed saprophytically after decortication."
- (5) "The Beri-Beri poison is probably an alkaloid which is stable and non-volatile and resembles atropine in some and muscarine in other of its effects."

DUBRUEL⁵ in a monograph "Le Béri-béri" 1906, gives the result of his observations on the disease in French Indo-China, and concludes that Beri-Beri is due to a pathogenic microbe developing in white rice which has been decorticated for some time. He believes that this pathogenic microbe taken with the rice produces an initial lesion in the alimentary canal, the other lesions characteristic of the disease following. He has not been able to isolate the supposed organism.

VAN DIK⁶ in a recent volume "Meelvergiftigen" calls attention to the points of similarity, clinical and pathological, displayed by Beri-Beri, Pellagra, Lathyrism and Ergotism and concludes that these diseases are forms of grain poisoning.

Numerous micro-organisms have been recorded as occurring in cases of Beri-Beri and by their discoverers have been held to be the causative agents. Satisfactory evidence in support of these claims has not yet been forthcoming. Certain other authorities while admitting that there is no proof that the disease is bacterial or protozoal in origin hold that the balance of evidence is in favour of such a view.

Rice.

Rice although not strictly a bread grain furnishes daily food for more human beings than any other cereal. It is the chief product in China, Indo-China, Japan, and other oriental countries. In the Federated Malay States and the Straits Settlements the amount of rice grown is not great. Its cultivation is for the most part confined to Malays and save in Perak and Province Wellesley the amount grown only suffices for the needs of the cultivators, who store the grain in bins and husk it as required.

To meet the requirement of a large and increasing number of immigrants, for the most part natives of China and India, a very large amount of rice must be imported annually.

Thus in 1907 the imports of padi and rice into the Straits Settlements were as follows:—

| COUNTRY OF ORIGIN. | PADI. | RICE. |
|----------------------|-------------|-----------|
| British North Borneo | ... 3,722 | ... |
| Burma Ports | ... 1,979 | 3,236,928 |
| Dutch Borneo | ... 10,568 | ... |
| French India | | 19,797 |
| French Indo-China | ... 18,823 | 759,519 |
| India | | 6,699 |
| Perak | ... 74,694 | ... |
| Siam | ... 43,668 | 3,420,755 |
| Siamese Malay States | ... 587,781 | 24,590 |
| Sumatra | ... 11,186 | 7,377 |

These figures indicate the quantities in pikuls. 1 pikul = 133½ pounds.

Penang and Singapore are largely ports of transhipment and from these ports a considerable amount of this grain is again exported to these States as well as to adjacent countries.

Padi, that is the grains of the rice plant, consists of the fruit enclosed in the paleæ which constitute the husk. The fruit possesses a thin pericarp firmly adherent to the seed and either silver-like or varying from dark red to black in colour. At the base of the dorsal edge may be seen the embryo lying in a depression. Subjacent to the pericarp are the spermoderm and the perisperm which surrounds the endosperm. The cells in the outer layers of the endosperm are filled with aleurone grains and the central portion of the seed is made up of cells closely packed with starch grains. Fat is practically confined to the outer layers of the seed.

In the preparation of rice from padi the product varies in accordance with the methods employed.

Rice as prepared by Malays.—Malays, who grow padi, employ primitive methods, they pound the grains until the husks are detached and remove them by winnowing. Portions of the pericarp at least and perhaps all of it are removed; should the pericarp be coloured it is almost certain to be wholly removed especially so in the case of careful housewives to whom this work is relegated and whose chief desire is to prepare a white rice. The layers subjacent to the pericarp are not interfered with to the same extent as when the grains are milled and polished by machinery.

White Rice.—The imported rice, other than that from India and Sumatra, may be described as white rice and is the kind preferred by the immigrant peoples, other than Tamils, as well as by the Malays resident in towns. In accordance with the market requirements the imported padi is milled either into this rice or parboiled rice.

White rice is the "stale uncured" rice of BRADDON and is the variety believed by him to be the source of the causative agent of Beri-Beri.

For the preparation of this form of rice large mills have been established in the places from which the rice is exported, also in Penang

and Singapore. In these mills the padi is husked and polished by machinery and since the demand is for a white rice polishing is carried out so thoroughly that in addition to the removal of the husk, pericarp, spermoderm and perisperm, a portion of the endosperm is removed; in consequence, the finished product consists almost wholly of cells closely packed with starch grains.

Parboiled Rice.—The Tamil labourer prefers a rice similar to that consumed by him in India and a small amount of such rice is imported from India and Sumatra but as the cost is considerably greater than that of white rice its use is limited to the more wealthy Tamils and to some institutions.

To meet the demand for a similar rice and to supply it at cheaper rates, padi is imported from the Siamese Malay States, Perak, and Province Wellesley and specially prepared in the mills of Penang and Singapore. The padi is placed in large concrete tanks and covered with water in which it is soaked for forty-eight hours or thereabouts. The moist padi is then transferred to lightly covered cylinders through which steam is passed for about five minutes. The padi is thereafter removed to paved courts and dried by exposure to the sun. It is then either stored as padi or milled at once. The milling and polishing process is identical with that employed in the preparation of white rice but the endosperm is not interfered with to the same extent and consequently the aleurone layer is for the most part preserved. In microscopic sections of parboiled rice fragments of the pericarp may be seen.

As compared with similar rices imported from India and Sumatra the local product has a peculiar disagreeable odour which can to some extent be removed by careful washing previous to cooking. The exact cause of this difference has not been determined. These rices are here called parboiled rice (the "cured" rice of BRADDON), the grains are of a yellowish colour and more or less translucent, it may be that as such rice cannot be made white the polishing is carried to a less degree, or the treatment previous to milling may have rendered the peripheral layers less friable and thus less readily removed by polishing.

Comparison microscopically of the Malay, parboiled and white rices, shows that the latter has been deprived to the greatest extent of its oil-containing and aleurone layers and chemically we have shown that parboiled rice contains relatively much more oil than white rice.

The investigation hereinafter described was undertaken primarily to determine if, when other factors were excluded or controlled people fed on white rice did develop Beri-Beri and if people under exactly similar conditions but fed on parboiled rice did not develop the disease. It was hoped also that opportunity would be forthcoming for the investigation of other aspects of the question.

At the outset it is necessary to state that the disease under investigation is that form of multiple peripheral neuritis, known as Beri-Beri, which occurs endemically in this peninsula and the neighbouring islands. As much confusion has been caused by assigning this name to classes of cases

differing widely in their clinical manifestations it is desirable to make it clear that we seek only for an explanation of this disease as met with here.

For the purpose of the inquiry it was necessary to observe two parties of men under similar conditions as to environment, etc., and whose food supply was definitely known. In view of the suggestion that the disease may be bacterial or protozoal in origin it was desirable that the places chosen should have been hitherto uninhabited or that no case of Beri-Beri should have occurred there for some time previously; further the places should be in an isolated district sufficiently remote from towns or villages to exclude as far as possible the entrance of a supposed infection. Such a situation would also have the advantage, on account of the absence of shops, that the men under observation could not readily obtain food other than that supplied to them. It is obvious that the conditions required for such an investigation could not be secured in a public institution as in all such in these States Beri-Beri is known to be endemic.

Various places were visited with a view to securing satisfactory conditions, and it was finally decided to carry on observations with regard to some three hundred Javanese indentured labourers employed in the work of road construction in a remote part of the Jelebu district in the State of Negri Sembilan. As will appear from the map the places in which the labourers were at this time located, Kuala Ayer Baning and Durien Tipus, were sufficiently remote from the nearest village or town for the purpose; Malay settlements in the district were few in number and small in size. In connection with these latter it should be remembered that abundant evidence exists to show that Malays in such situations do not suffer from Beri-Beri.

The first indentured Javanese employed on road construction in the Jelebu district commenced work at Pesasi in March 1905. At that time only a small number of Javanese were employed and during the year following they were moved to Pertang. In September 1906, the work having been completed, many of the labourers were sent further along the road and occupied quarters newly erected at Kuala Ayer Baning (51½ mile) and in January 1907 quarters newly erected were occupied at Durien Tipus (58th mile). By the end of 1906 the number of Javanese employed had been largely increased and then numbered about four hundred.

Under the terms of contract the rice issued to these labourers was supplied by the contractor and as they prefer white rice this kind was supplied. In the early months of 1906 Beri-Beri occurred among them and in May, June and July of that year it was a serious source of invaliding and mortality. From August 2nd, 1906, the employer, adopting the suggestion of Dr. BRADDON, issued only parboiled rice instead of white rice; thenceforward it is stated, and this statement is confirmed by the hospital records, no case of Beri-Beri occurred.

Here then the conditions seemed to be in every way suitable for an inquiry into the part played by rice in the causation of Beri-Beri because these labourers without exception still desired to return to a white rice diet and at this time the evidence of a connection between the consumption of

white rice and Beri-Beri was by no means convincing either to the general body of medical and scientific workers or to ourselves. The importance of reaching some conclusion regarding the origin of the disease cannot be over-estimated as the number of its victims in this Peninsula alone runs into many thousands annually.

Throughout these States no labourers other than Tamils will consume parboiled rice unless compelled to do so and while there was any doubt as to the harmful influence of white rice no effective measures could be taken for the suppression of Beri-Beri.

By acceding to the wishes of the group of labourers comprised in this investigation opportunity would be afforded for a thorough testing of the position of dietary factors as causative agents. The labourers were therefore given the option of returning to a white rice diet after it had been fully explained to them that by so doing they ran the risk of contracting Beri-Beri. Without exception they chose the white rice but as for the purpose of comparison two parties were required, half the number only were allowed this diet. It was hoped also that by continuous observation of a large party of men on a parboiled rice diet it might be determined whether, apart from its disagreeable musty odour, any grounds existed for the objections made to the consumption of this rice.

At the time the investigation was commenced, April 1907, the three hundred labourers then remaining were divided into two parties of approximately equal numbers, the one party at Kuala Ayer Baniang, the other party at Durien Tipus. The clearings for the quarters had been made in virgin jungle and no case of Beri-Beri had occurred at either place. The quarters were well raised from the ground, the floors made of split bamboo, the walls of bark, and the roof of light attaps, thus they were well ventilated. In all cases the lines were well drained and near running water. The sanitary conditions were good.

In April all the labourers were examined and found to be free from any sign of existing or recent Beri-Beri. The results of the physical examination of each person were recorded for future reference and an arrangement made that any person subsequently joining the parties should be carefully examined previously.

An interval was allowed to elapse during which any latent case might be expected to develop and as all remained healthy white rice was issued to the Durien Tipus party for the first time on May 12th, the Kuala Ayer Baniang party remaining on parboiled rice as before. *The rices were of uniformly good quality and were obtained in quantities sufficient for one month at a time.*

DIET.

The daily ration was as follows:-

| | | |
|-----------------|------|------|
| Rice | 21.3 | OZS. |
| Dried Salt Fish | 4.25 | " |
| Onions | 1.75 | " |
| Potatoes | 1.75 | " |
| Coconut Oil | 0.85 | " |
| Coconut | 1.50 | " |
| Tea | 0.12 | " |
| Salt | 0.1 | " |

The various articles composing this diet were submitted to analysis in this Institute by Mr. EATON, and the results obtained were as follows:—

White Rice—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|-----------|-------|----------------|--------|
| 7.45 | 0.17 | 78.02 | 0.51 |

Parboiled Rice—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|-----------|-------|----------------|--------|
| 7.8 | 0.53 | 76.92 | 0.72. |

Dried salt fish—

The species of fish so prepared are numerous, and the kind issued to the coolies varied to some extent. The average results from the analyses of a number of these species were—

| Proteids. | Fats. | Salts. |
|-----------|-------|--------|
| 35.7 | 2.96 | 13.24 |

Onions—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|-----------|-------|----------------|--------|
| 1.5 | 0.28 | 11.28 | 0.5 |

Potatoes—

| Proteids. | Fats | Carbohydrates. | Salts. |
|-----------|------|----------------|--------|
| 1.8 | 0.2 | 31.66 | 1.04 |

Coconuts—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|-----------|-------|----------------|--------|
| 3.95 | 34.6 | 15.2 | 0.95 |

Based on these analyses the diet issued to those consuming white rice has been calculated to consist of—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|-----------------|--------------|---------------------|--------------|
| 91.451 grms. | 43.708 grms. | 499.165 grms. | 23.064 grms. |
| and to contain— | | Carbon 303.75 grms. | |
| | | Nitrogen 14.8 „ | |

The diet issued to those consuming parboiled rice has been calculated to consist of—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|----------------|--------------|---------------------|--------------|
| 93.565 grms. | 45.882 grms. | 492.540 grms. | 24.335 grms. |
| and to contain | | Carbon 304.34 grms. | |
| | | Nitrogen 15 „ | |

In accordance with European standards it is stated that 2 grammes of proteid, 1.5 grammes of fat, 6 grammes of carbohydrate, and 0.5 gramme of salt per kilogramme of body-weight are required. The average body-weight of the persons under observation was about 100 lbs. and for an individual of that weight therefore the diet should contain—

| Proteids. | Fats. | Carbohydrates. | Salts. |
|-------------------|----------|---------------------|----------|
| 90.72 grms. | 68 grms. | 272.16 grms. | 20 grms. |
| and would contain | | Carbon 220.73 grms. | |
| | | Nitrogen 14.6 „ | |

If now this calculated standard diet be compared with the diets consumed by the persons under observation it will be seen that *the issued diets can in no way be regarded as deficient*. It is true that the amounts of fat are considerably less than in the standard diet but the deficiency in this respect is more than compensated for by the excess of carbohydrate.

Nearly one-half of the proteid material in the diets issued is derived from rice, and it may be suggested that the treatment of the grain previous to milling renders the proteid of parboiled rice more readily available than that of white rice. So far as a limited number of experiments have gone no evidence has been obtained in support of this suggestion.

It is well known that with a sufficiency of proteid in any diet, fats and carbohydrates are to a considerable extent interchangeable and it must not be forgotten that the inhabitants of warm countries habitually consume relatively less fat and more carbohydrate than do the inhabitants of cold countries.

The parties were under constant medical supervision throughout the course of the inquiry and while as already stated the investigation was primarily directed to the part played by rice in the causation of Beri-Beri, attention was also directed to other factors which have been suggested as bearing upon its etiology. Thus the incidence of *ankylostomiasis*, malaria, etc., was ascertained and a general examination of all the coolies made at intervals of about one month and more frequent examinations of those showing signs of illness. In these examinations attention was particularly directed to the nervous system so that in the event of Beri-Beri developing there should be a complete record of the patient's condition during the preceding months.

The parties as originally formed are designated Party No. I (Durien Tipus); Party No. II (Kuala Ayer Bening), and Party No. III, a small party at Pertang.

On July 1st the requirements of the work necessitated the division of Party No. I into two groups. One group of approximately fifty (Party No. I A.) remained at Durien Tipus, the other group, about one hundred in number (Party No. I B.), were transferred to Juntai at the 56th mile. The quarters at this latter place had been newly erected in a fresh clearing. The conditions as regards food remained unchanged. The individuals in these two groups being under similar dietary conditions were, for the purpose of this investigation, regarded as one party and were moved freely from one place to the other.

In order to enable the history of each individual to be followed both as to place of residence and as to diet, we have made use of Tables. In these the individual retains the same number throughout. Red and black lines indicate the period of time each individual was on white or parboiled rice respectively. If not a member of the party as originally formed the place from whence he came is indicated at the beginning of the line and if he left the party before the conclusion of the investigation the place to which he went is also indicated; the names of these places are shown in red or black according as the parties were on white or parboiled rice.

PARTY No. I. (*Durian Tipus*)

May—June, 1907.

PARTY No. I. A. (*Durian Tipus*)

July—October, 1907.

| | MAY | JUNE | JULY | AUGUST | SEPTEMBER | OCTOBER |
|----|-------|-------|-------------------|--------|---------------------|---------|
| 1 | _____ | _____ | Party No. I. B. . | _____ | _____ | _____ |
| 2 | _____ | _____ | " " | | | |
| 3 | _____ | _____ | " " | | | |
| 4 | _____ | _____ | _____ | Left | | |
| 5 | _____ | _____ | Party No. I. B. | | | |
| 6 | _____ | _____ | _____ | | | |
| 7 | _____ | _____ | Party No. I. B. | | | |
| 8 | _____ | _____ | " " | | | |
| 9 | _____ | _____ | B.B. | | | |
| 10 | _____ | _____ | Party No. I. B. | | | |
| 11 | _____ | _____ | " " | | | |
| 12 | _____ | _____ | " " | | | |
| 13 | _____ | _____ | _____ | | | |
| 14 | _____ | _____ | Party No. I. B. | | | |
| 15 | _____ | _____ | " " | | | |
| 16 | _____ | _____ | " " | | | |
| 17 | _____ | _____ | " " | | | |
| 18 | _____ | _____ | " " | | | |
| 19 | _____ | _____ | " " | | | |
| 20 | _____ | _____ | " " | | | |
| 21 | _____ | _____ | " " | | | |
| 22 | _____ | _____ | " " | | | |
| 23 | _____ | _____ | Party No. I. B. | _____ | | |
| 24 | _____ | _____ | _____ | | | |
| 25 | _____ | _____ | _____ | | | |
| 26 | _____ | _____ | Party No. I. B. | | | |
| 27 | _____ | _____ | Party No. I. B. | _____ | Died.
Pneumonia. | |
| 28 | _____ | _____ | " " | | | |
| 29 | _____ | _____ | " " | | | |

| | MAY | JUNE | JULY | AUGUST | SEPTEMBER | OCTOBER |
|----|-----|------|-----------------|--------|-----------|---------|
| 30 | | | Party No. I. B. | | | |
| 31 | | | " | " | | |
| 32 | | | " | " | | |
| 33 | | | " | " | | |
| 34 | | | " | " | | |
| 35 | | | " | " | | |
| 36 | | | Party No. I. B. | | | |
| 37 | | | | | B.B. | |
| 38 | | | Party No. I. B. | | | |
| 39 | | | " | " | | |
| 40 | | | " | " | | |
| 41 | | | " | " | | |
| 42 | | | Hospital | | Hospital | |
| 43 | | | | | | |
| 44 | | | Party No. I. B. | | | |
| 45 | | | " | " | | |
| 46 | | | " | " | | |
| 47 | | | " | " | | |
| 48 | | | " | " | | |
| 49 | | | " | " | | |
| 50 | | | " | " | | |
| 51 | | | Party No. I. B. | | | |
| 52 | | | " | " | | |
| 53 | | | " | " | | |
| 54 | | | " | " | | |
| 55 | | | " | " | | |
| 56 | | | " | " | B.B. | |
| 57 | | | " | " | | |
| 58 | | | " | " | | |
| 59 | | | " | " | | |
| 60 | | | " | " | | |
| 61 | | | " | " | | B.B. |

| | MAY | JUNE | JULY | AUGUST | SEPTEMBER | OCTOBER |
|----|-----|----------|-----------------|-----------------|----------------|-----------------|
| 62 | | | | | | |
| 63 | | | Party No. I. B. | | | |
| 64 | | | " " | | | |
| 65 | | | | | | Died. Epilepsy. |
| 66 | | | Party No. I. B. | | | |
| 67 | | | | B.B. | | |
| 68 | | | | | | |
| 69 | | | Party No. I. B. | | | |
| 70 | | | | | | |
| 71 | | | | | | |
| 72 | | | Party No. I. B. | | | |
| 73 | | | " " | | | |
| 74 | | | " " | | | |
| 75 | | | " " | | | |
| 76 | | | " " | | | |
| 77 | | | | | Party No. III. | |
| 78 | | | | | | |
| 79 | | | | Party No. I. B. | | |
| 80 | | | | " " | | |
| 81 | | | | " " | | |
| 82 | | | | " " | | |
| 83 | | | | " " | | |
| 84 | | | | " " | | |
| 85 | | | | " " | | |
| 86 | | Hospital | | " " | | |
| 87 | | | | " " | | |
| 88 | | | | " " | | |
| 89 | | | | " " | | |
| 90 | | | | " " | | |
| 91 | | | Party No. I. B. | From K. Pilah | | |
| 92 | | | " " | | | |
| 93 | | | | | | |

MAY JUNE JULY AUGUST SEPTEMBER OCTOBER

| | | |
|-----|------------------|-----------------------------|
| 94 | _____ | Died. Traumatic Meningitis. |
| 95 | _____ | Party No. I. B. |
| 96 | _____ | " " |
| 97 | _____ | " " |
| 98 | _____ | " " |
| 99 | _____ | " " |
| 100 | _____ | " " _____ Left |
| 101 | _____ | " " |
| 102 | From gaol _____ | " " |
| 103 | _____ | " " |
| 104 | _____ | " " |
| 105 | _____ | " " |
| 106 | _____ | " " |
| 107 | _____ | " " |
| 108 | _____ | " " |
| 109 | _____ | " " |
| 110 | _____ | " " |
| 111 | _____ | " " |
| 112 | _____ | " " |
| 113 | _____ | " " |
| 114 | _____ | " " |
| 115 | _____ | " " |
| 116 | _____ | " " |
| 117 | _____ | " " |
| 118 | _____ | " " |
| 119 | _____ | |
| 120 | _____ | Party No. I. B. |
| 121 | _____ | " " |
| 122 | _____ | " " _____ |
| 123 | From Hosp. _____ | " " |
| 124 | Hosp. _____ | " " _____ B.B. |
| 125 | " _____ | |

MAY JUNE JULY AUGUST SEPTEMBER OCTOBER

| | | | | |
|-----|---------------|---|----------------------|-----------------|
| 126 | | | | |
| 127 | From Pertang. | | | B.B. |
| 128 | Hosp. | | Party No. I. B. | Hosp. |
| 129 | Gaol | | | |
| 130 | | | | |
| 131 | | | | |
| 132 | | | From Hospital | |
| 300 | | | From Party No. I. B. | |
| 308 | | | From Kuala Pilah | |
| 321 | | | From Party No. II. | |
| 336 | | | From Party No. I. B. | Left |
| 340 | | | From Party No. II. | Left |
| 345 | " | " | " | |
| 346 | " | " | " | |
| 373 | " | " | " | Party No. I. B. |
| 375 | | | From Party No. I. B. | |
| 376 | | | From Party No. I. B. | |
| 389 | | | From Party No. II. | |

PARTY No. 1 (May—June, 1907).

As will appear from the table Party No. 1 comprised those individuals who were on white rice at Durien Tipus from May 12th until July 1st when the party was divided into two groups.

The history of Party No. 1 calls for no special comment. No case of Beri-Beri occurred in it during the period May 12th to July 1st.

Dealing now with the question of food as a source of the disease it will be seen that Party No. I A. were on white rice from May 12th until October 11th. During this period thirty members of this party were on white rice for three months or longer and amongst these seven cases of Beri-Beri occurred. During the time Beri-Beri was present at Durien Tipus, August 7th to October 11th seven individuals joined the party, either from Party No. II (Kuala Ayer Bening) or from hospital, there was no white rice issued at these places and none of these seven developed Beri-Beri though they were exposed to the chances of an infection equally with the other members of the party. Of thirteen individuals who came from Party No. I B. where white rice was being issued two subsequently contracted Beri-Beri.

The results in this party therefore suggested the possibility that a diet of which white rice formed the staple was in some way concerned in the production of Beri-Beri.

PARTY No. I. B. (*Funtai*).

May to October, 1907.

| | MAY | JUNE | JULY | AUGUST | SEPTEMBER | OCTOBER |
|----|-----|-------------|------|--------|-----------|-----------------|
| 1 | | Party No I. | | | | Party No. I. A. |
| 2 | | " " | | | | |
| 3 | | " " | | | | |
| 5 | | " " | | | | Left |
| 7 | | " " | | | | B.B |
| 8 | | " " | | | | |
| 10 | | " " | | | | |
| 11 | | " " | | | | |
| 12 | | " " | | | | |
| 14 | | " " | | | | |
| 15 | | " " | | | B.B. | |
| 16 | | " " | | | | |
| 17 | | " " | | | | Died. |
| 18 | | " " | | | | |
| 19 | | " " | | | | |
| 20 | | " " | | | | |
| 21 | | " " | | | | |
| 22 | | " " | | | | Left |
| 23 | | " " | | | | Party No. I. A. |
| 26 | | " " | | | | |
| 27 | | " " | | | | Party No. I. A. |
| 28 | | " " | | | | |
| 29 | | " " | | | | |

| | MAY | JUNE | JULY | AUGUST | SEPTEMBER | OCTOBER |
|----|-----|--------------|------|-----------------|-----------|-----------------|
| 30 | | Party No. I. | | | | |
| 31 | | " " | | | | |
| 32 | | " " | | | | |
| 33 | | " " | | | | Left |
| 34 | | " " | | | | Left |
| 35 | | " " | | | | |
| 36 | | " " | | Party No. I. A. | | |
| 38 | | " " | | | | |
| 39 | | " " | | | | |
| 40 | | " " | | | | |
| 41 | | " " | | | | |
| 44 | | " " | | | | Left |
| 45 | | " " | | | | Party No. I. A. |
| 46 | | " " | | | | |
| 47 | | " " | | | | Left |
| 48 | | " " | | | | |
| 49 | | " " | | | | Party No. I. A. |
| 50 | | " " | | | | |
| 51 | | " " | | Party No. I. A. | | |
| 52 | | " " | | | | |
| 53 | | " " | | | | Party No. I |
| 54 | | " " | | | | |
| 55 | | " " | | | | |
| 56 | | " " | | Party No. I. A. | | |
| 57 | | " " | | | | B.B. |
| 58 | | " " | | | | Left |
| 59 | | " " | | | | |
| 60 | | " " | | | | Party No. I. A. |
| 61 | | " " | | | | |

| | | |
|----|---------------|-----------------|
| 63 | Party No. I. | |
| 64 | " " | Hosp. |
| 66 | " " | |
| 69 | " " | |
| 72 | " " | Party No. I. A. |
| 73 | " " | Party No. I. A. |
| 74 | " " | |
| 75 | " " | |
| 76 | " " | B.B. |
| 79 | " " | |
| 80 | " " | |
| 81 | " " | |
| 82 | " " | |
| 83 | " " | Party No. I. A. |
| 84 | " " | |
| 85 | " " | |
| 86 | From Hospital | Party No. I. |
| 87 | Party No. I. | |
| 88 | " " | |
| 89 | " " | Hosp. |
| 90 | " " | |
| 91 | " " | Absconded |
| 92 | " " | |

MAY

JUNE

JULY

AUGUST

SEPTEMBER

OCTOBER

95

Party No. 1

96

. .

97

. .

98

. .

99

. .

100

. .

Party No. I. A.

101

. .

102

. .

103

. .

104

. .

105

. .

106

. .

Left

107

. .

108

. .

109

. .

B.B.

110

. .

111

. .

112

. .

113

. .

114

. .

115

. .

116

. .

117

. .

118

. .

120

. .

121

. .

122

. .

Party No. I. A.

123

. .

124

. .

Party No. I. A.

MAY

JUNE

JULY

AUGUST

SEPTEMBER

OCTOBER

| | | |
|-----|--------------|-----------------------|
| 128 | Party No. I. | _____ Party No. I. A. |
| 129 | " " | _____ |
| 130 | " " | _____ Party No. II. |
| 131 | " " | _____ " " |

| | | | |
|-----|--|--------------------|-----------------------|
| 300 | | From Party No. II. | _____ Party No. I. A. |
| 306 | | From Party No. II. | _____ Party No. I. A. |
| 375 | | " " " | _____ " " |
| 376 | | From Party No. II. | _____ |
| 393 | | From Party No. II. | _____ |
| 421 | | From Party No. II. | _____ |

*** PARTY No. I B.**

The first case of Beri-Beri (No. 15) to develop in this party was taken ill on the 29th of September. He had been in the party since its formation on July 1st and had been on white rice in all 141 days.

The second case (No. 7) developed on October 10th. This man had been on white rice 152 days.

The third, fourth and fifth cases Nos. 76, 57, and 109, followed rapidly, the dates being October 12th, 16th and 18th. These cases had been on white rice 134, 158 and 160 days respectively.

From the view point of infection there is little to be said regarding this party. Such may easily have been introduced from Party No. I A at Durien Tipus where the disease had broken out seven weeks previously. These two parties were located only two miles apart and on holidays, which occurred twice a month, very slight restraint was placed upon their movements as for the primary purpose of this inquiry they were regarded as one party.

The introductions from outside into this party were No. 86 who returned from Kuala Klawang Hospital on July 12th. No. 102 who joined Party No. I from Kuala Klawang gaol on June 22nd and was transferred to Party No. I B. on its formation, July 1st. Five individuals were transferred to this party from Party No. I A. on July 23rd after which date no other transfers were made. Six people were moved from Party No. II to this party. None of the individuals here referred to developed Beri-Beri.

It is proper to mention here that in determining whether a given case was to be admitted as a case of Beri-Beri the most rigid exclusion was practised. Only such cases as presented unequivocal signs of the disease were admitted. In every instance the diagnosis was based on the opinion of at least two medical men, in most instances by that of four. Where any doubt was cast upon the accuracy of the diagnosis such case was rejected. The result therefore is that, apart from the cases here recorded, there were many others which, in the opinion of the writers as well as of those associated with them in this inquiry, were really mild or obscure cases of the disease. The difficulties in this respect will be appreciated by those who have had to deal with the disease clinically. No such doubtful case was at any time observed among the people on parboiled rice, and the inclusion of cases of this type occurring in the white rice parties does in no way strengthen the case for an infectious origin of the disease.

By October 11th seven cases had occurred in Party No. I A. and by October 18th five cases in Party No. I B. As there was apparently nothing further to be gained from a continuance of the white rice diet, it was thought that the time was suitable by a change to parboiled rice to observe the effect of this alteration of diet upon the course of the outbreak. Accordingly parboiled rice was substituted for white rice in the diet of Party No. I A. on October 12th and in that of Party No. I B. on October 19th.

After this change no case of Beri-Beri occurred in either party and such cases as showed signs suspicious of Beri-Beri rapidly got well.

This abrupt cessation of the outbreak constitutes important evidence of a causative relationship between the consumption of white rice and Beri-Beri. It is known that diseases of infectious origin do tend to die out abruptly and it is admitted that this cessation is not incompatible with an hypothesis of an infectious origin for the disease and it is possible that the cessation of the outbreak on the change of rice was merely a coincidence and that all the susceptible individuals had developed Beri-Beri.

The results in Parties No. I A. and No. I B. may be tabulated as follows:

I. Assuming an infectious origin for Beri-Beri.

PARTY No. I A.

| | | |
|------|-----|--|
| Case | I | August 6th. |
| " | II | " 19th. |
| " | III | September 3rd (exposed to infection August 23rd). |
| " | IV | " 6th (directly exposed to infection August 23rd). |
| " | V | " 18th. |
| " | VI | October 5th (transferred from Party No. I B. October 1st). |
| " | VII | " 10th. |

PARTY No. I B.

| | | |
|------|-----|-----------------|
| Case | I | September 29th. |
| " | II | October 10th. |
| " | III | " 12th. |
| " | IV | " 16th. |
| " | V | " 18th. |

II. Assuming a dietary origin.

| | | | | | | | |
|------|-------|------------------------------|-----|---|---|-----|---|
| | Up to | 30 days on white rice, cases | | | | ... | 0 |
| From | 31 | „ | 60 | „ | „ | „ | 0 |
| „ | 61 | „ | 90 | „ | „ | „ | 1 |
| „ | 91 | „ | 120 | „ | „ | „ | 4 |
| „ | 121 | „ | 150 | „ | „ | „ | 3 |
| „ | 151 | „ | 160 | „ | „ | „ | 4 |

PARTY No. II. (*Kuala Ayer Buning*).

May—October, 1907.

MAY

JUNE

JULY

AUGUST

SEPTEMBER

OCTOBER

130

131

300 _____ Party No. I. B.

301 _____

302 _____ Hospital _____

303 _____

304 _____

305 _____

306 _____

307 _____ Hosp. _____

308 _____ Absconded

309 _____ Hosp. _____

310 _____

311 _____ Hosp. _____

312 _____

313 _____

314 _____

MAY

JUNE

JULY

AUGUST

SEPTEMBER

OCTOBER

| | | | | | |
|-----|--|----------|-------|-----------------|-------|
| 315 | | | | | |
| 316 | | | | | |
| 317 | | | | Left | |
| 318 | | | | | |
| 319 | | | | | |
| 320 | | | | Absconded | |
| 321 | | | | Party No. I. A. | |
| 322 | | | Hosp. | | |
| 323 | | | | Left | |
| 324 | | | | Left | |
| 325 | | | | | |
| 326 | | | | | Hosp. |
| 327 | | | | | |
| 328 | | | | | |
| 329 | | | | | |
| 330 | | | | Left | |
| 331 | | | | | |
| 332 | | | | | |
| 333 | | Gaol | | Left | |
| 334 | | | | Left | |
| 335 | | | | | |
| 336 | | | | Party No. I. B. | |
| 337 | | | | | |
| 338 | | | | | |
| 339 | | | | Left | |
| 340 | | | | Party No. I. A. | |
| 341 | | | | | |
| 342 | | | | | |
| 343 | | | | | |
| 344 | | | | | |
| 345 | | Hospital | | Party No. I. A. | |
| 346 | | | | " " | |

MAY JUNE JULY AUGUST SEPTEMBER OCTOBER

| | | | | | |
|-----|--|----------|--|------------------|-----------------|
| 347 | | | | | |
| 348 | | | | | |
| 349 | | | | | |
| 350 | | | | Left | |
| 351 | | | | | |
| 352 | | | | | Hosp. |
| 353 | | | | | |
| 354 | | | | Hospital | |
| 355 | | Hospital | | | |
| 356 | | | | | |
| 357 | | | | | |
| 358 | | | | Died. Dysentery. | |
| 359 | | | | Hospital | |
| 360 | | | | | |
| 361 | | | | | |
| 362 | | | | | |
| 363 | | | | | |
| 364 | | | | | |
| 365 | | | | | |
| 366 | | | | Hospital | |
| 367 | | | | | |
| 368 | | | | | |
| 369 | | | | | |
| 370 | | | | | |
| 371 | | | | | |
| 372 | | | | | |
| 373 | | | | Party No. I. A. | |
| 374 | | | | | |
| 375 | | | | Party No. I. B. | |
| 376 | | | | | Party No. I. B. |
| 377 | | | | | Hosp. |
| 378 | | | | | |

MAY

JUNE

JULY

AUGUST

SEPTEMBER

OCTOBER

| | | | | | | |
|-----|--|--|--|------------------|------|--|
| 379 | | | | | | |
| 380 | | | | | Left | |
| 381 | | | | | | |
| 382 | | | | | | |
| 383 | | | | | | |
| 384 | | | | | Left | |
| 385 | | | | | | |
| 386 | | | | | | |
| 387 | | | | | | |
| 388 | | | | | | |
| 389 | | | | Party No. I. A. | | |
| 390 | | | | | | |
| 391 | | | | | | |
| 392 | | | | | | |
| 393 | | | | Party No. I. B. | | |
| 394 | | | | | | |
| 395 | | | | | | |
| 396 | | | | | | |
| 397 | | | | | | |
| 398 | | | | | | |
| 399 | | | | | | |
| 400 | | | | | | |
| 401 | | | | | | |
| 402 | | | | | | |
| 403 | | | | | | |
| 404 | | | | | | |
| 405 | | | | | | |
| 406 | | | | | | |
| 407 | | | | | | |
| 408 | | | | Left | | |
| 409 | | | | | | |
| 410 | | | | Died. Dysentery. | | |

| | MAY | JUNE | JULY | AUGUST | SEPTEMBER | OCTOBER |
|-----|----------|------|-------|----------|-----------------|---------|
| 411 | | | | | | |
| 412 | | | | | | |
| 413 | | | | Hospital | | |
| 414 | | | | | | |
| 415 | | | | | | |
| 416 | | | | | | |
| 417 | | | | | | |
| 418 | | | | | | |
| 419 | | | | | | |
| 420 | | | | | | |
| 421 | | | | | Party No. I. B. | |
| 422 | | | | | | |
| 423 | | | | | | |
| 424 | | | | | | |
| 425 | | | | | Hospital | |
| 426 | Hospital | | | Hospital | | |
| 427 | " | | | | | |
| 428 | " | | Hosp. | | | |
| 429 | " | | | Left | | |
| 430 | " | | | | | |
| 431 | | | | | | |
| 432 | | | | | | |
| 433 | | | | | | |
| 434 | | Gaol | | | | |
| 435 | | | | | | |
| 436 | | | | | Hospital | |
| 437 | Hospital | | | | | |
| 438 | | | | Hospital | | |
| 439 | | | | Gaol | | |
| 440 | | | Gaol | | | |

PARTY No. II (May—October, 1907).

This party located at Kuala Ayer Baniang was employed for purposes of comparison with the results in Parties No. I A. and No. I B. At this place only parboiled rice had been issued since the quarters were first occupied in September 1906. These conditions were continued till October, 1907, and the party was under observation from May till October. The same care was taken in examining the coolies comprising this party as was taken in dealing with members of the white rice parties.

At one time or another one hundred and forty-three individuals were members of this party. Of these one hundred and twelve were on parboiled rice continuously from May 1st until the date (August 7th) when Beri-Beri first appeared in Party No. I A. and eighty-nine remained on parboiled rice until the change to white rice on October 19th. There were present in the party for at least three months one hundred and twenty-one and there were present during the period August 7th—October 19th one hundred and thirty-one.

During the period, May 1st—October 19th, men left the party to go to hospital and some two or three were sent to gaol. The majority of these returned to the party on their recovery or release. Of such returns there were in all twenty-five, twenty-one from hospital and four from gaol, who may be regarded as potential sources or carriers of an infection in contrast to thirteen who joined Parties No. I A. and No. I B. during the same period. In addition, this party was located nearer a settlement than either Party No. I A. or Party No. I B. It will thus appear that the chances for the introduction of an hypothetical infection were much greater in the case of the control Party No. II than in Parties No. I A. and No. I B. Despite this preponderance of factors favouring the introduction of infection no case of Beri-Beri appeared in this group.

PARTY No. II (October, 1907—May, 1908).

In view of the results obtained, it was decided to reverse the conditions and to place this party, hitherto on parboiled rice, on white rice, and to have Parties No. I A. and No. I B. henceforward on parboiled rice. This change was made in Party No. II on October 19th, no other alteration, dietetic or otherwise, being made.

The better to safeguard this party from infection it was decided that, so far as it was possible to do so, no one should be allowed to join the party from outside. All persons sent to hospital were on their return assigned to other parties. In consequence of this procedure the numbers of this party materially diminished during the course of the investigation and gradually it came to be a more or less selected party, only the more robust of its members remaining.

On the 19th of October there were at Kuala Ayer Baning one hundred and eleven people a number of whom left or were transferred soon after that date. The removal of the party from Kuala Ayer Baning took place on March 22nd, they were therefore under observation at that place for one hundred and sixty-five days. Fifty-eight of the party had been continuously present since the 19th of October and one from the 28th of October. Five men joined the party from hospital on the 2nd of October and one joined on the 28th of October. One other joined from hospital on the 13th November. Thenceforward as already stated no one was allowed to join this party.

This period of one hundred and sixty-five days at Kuala Ayer Baning considerably exceeded the minimum interval which elapsed at either Juntai or Durien Tipus between the first issue of white rice and the outbreak of Beri-Beri. Seventy-seven coolies were under observation for at least ninety days.

As the work of road construction had been completed at Kuala Ayer Baning by the 22nd of March, the labourers, fifty-four in number, comprising the party on that date were transferred to new quarters at the 64th mile. These quarters had been erected about two months previously and had not been occupied. Between the old and new quarters the party was not exposed to any possible infection so far as is known.

On April 9th No. 311 developed Beri-Beri and about the same time No. 371 also developed the disease. Owing to the requirements of the work it was found necessary on the 23rd of April to transfer the remaining members of the party to quarters at the 61½ mile. These quarters had been occupied since November 18th by Party No. I which comprised ninety to one hundred and ten persons and amongst whom no case of Beri-Beri had occurred since the change of rice on October 11th and 19th. The two cases of Beri-Beri which had occurred at the 64th mile were, as soon as the diagnosis was established, transferred to the quarters at 61½ mile in order to be more directly under medical care and as the remainder of the party were transferred on the date mentioned it is clear that abundant opportunity was afforded by this movement for the transference of an infection if such had existed.

PARTY No. II. (*Kuala Ayer Bening, 64th Mile, 61½ Mile*)

October 1907—May 1908.

| | OCT. | NOV. | DEC. | JAN. | FEB. | MARCH | APRIL | MAY |
|-----|------|--------------|-------------------|------|-------------------|-------|--------------|---------------|
| 46 | | | | | From Party No. I. | | | Died. Dysent. |
| 104 | | | | | " " | | | Gaol |
| 111 | | | | | " " | | | |
| 115 | | | | | " " | | | |
| 116 | | | | | " " | | | |
| 130 | | | | | | | | |
| 131 | | Party No. I. | | | | | Party No. I. | |
| 142 | | | | | From Party No. I. | | | |
| 146 | | | | | " " | | | |
| 148 | | | From Party No. I. | | | | Party No. I. | |
| 177 | | | | | From Party No. I. | | | |
| 301 | | | | | | | | |
| 302 | | | | | | | | |
| 303 | | | Hospital | | | | | |
| 304 | | | | | | | | |
| 305 | | | | | Party No. I. | | | |
| 306 | | | Hospital | | | | | |
| 307 | | | " " | | | | | |
| 309 | | | | | | | | B.B. |
| 310 | | | | | | | Left | |
| 311 | | | | | | | B.B. | Hosp. |
| 312 | | | | | | | | |
| 313 | | | | | | | | |
| 314 | | | | | | | Party No. I. | |

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

315 _____

316 _____ Party No. I.

318 _____ Left

319 _____

322 _____

325 _____ Hospital

326 Hosp. _____ Died

327 _____ Left

328 _____ Left

329 _____

331 _____

332 _____ Left

335 _____ Left

337 _____

338 _____ Party No. I.

341 _____

342 _____ Left

343 _____ Left

344 _____ Left

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

| | | | | | | | |
|-----|-------|--|--------------|--------------|--------------|--------------|-----------|
| 347 | | | | | | | B.B. |
| 348 | | | | | | | |
| 349 | | | Left | | | | |
| 351 | | | Left | | | | |
| 353 | | | | | | | |
| 354 | | | | | | | |
| 355 | | | Hospital | | | | |
| 356 | | | | | | | |
| 357 | | | | | | Party No. I. | |
| 359 | | | | | | | |
| 360 | | | | Party No. I. | | | |
| 361 | | | | | | Left | |
| 362 | | | | | | | |
| 363 | | | | | | | |
| 364 | | | Left | | | | |
| 365 | | | Left | | | | |
| 366 | | | | | Left | | |
| 367 | | | Party No. I. | | | | |
| 368 | | | Left | | | | |
| 369 | | | | Party No. I. | | | |
| 370 | | | | Party No. I. | | | |
| 371 | | | | | | B.B. | Died Dys. |
| 372 | | | | | Party No. I. | | |
| 374 | | | | | | | |
| 377 | Hosp. | | | | | Left | |
| 378 | | | | | | Left | |

379 _____

381 _____

382 _____ Party No. I.

383 _____ Party No. I.

385 _____

386 _____ Hosp.

387 _____ Party No. I.

388 _____ B.B.

390 _____ Left

391 _____ Left

392 _____ Left

394 _____ Left

395 _____ Party No. III.

396 _____ Left

397 _____ Party No. III.

398 _____

399 _____

400 _____

401 _____

402 _____

403 _____

404 _____

405 _____ B.B.

406 _____ Left

407 _____

409 _____ Left

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

| | | | |
|-----|-------|----------------|--------------------|
| 411 | _____ | Party No. III. | |
| 412 | _____ | Left | |
| 414 | _____ | Left | |
| 415 | _____ | " | |
| 416 | _____ | " | |
| 417 | _____ | " | |
| 418 | _____ | Party No. I. | |
| 419 | _____ | | |
| 420 | _____ | | |
| 422 | _____ | Party No. I. | |
| 423 | _____ | Left | |
| 424 | _____ | | Left |
| 425 | _____ | | Left |
| 426 | _____ | Left | |
| 427 | _____ | | |
| 428 | _____ | Left | |
| 430 | _____ | | Died. Dys. |
| 431 | _____ | | |
| 432 | _____ | Left | |
| 433 | _____ | | B.B. |
| 434 | _____ | Party No. I. | _____ Party No. I. |
| 435 | _____ | Left | |
| 436 | _____ | Hospital | |
| 437 | _____ | Left | |
| 438 | _____ | Hospital | |
| 439 | _____ | Left | |
| 440 | _____ | Party No. I. | |

When the change had been effected there were occupying the quarters at the $61\frac{1}{2}$ mile one hundred and thirty-one people, forty-two who had been and were now continued on white rice, eighty-five who had been and were now continued on parboiled rice, and four individuals who had been transferred from the 64th mile previous to the 23rd April. Two of these latter were cases of Beri-Beri and two were suspected cases.

The food for the mixed party other than the rice was prepared in a common kitchen; the rices were prepared in separate kitchens and in order to be quite certain that the people on white rice were not given parboiled rice or *vice versa* the food was issued to the parties at different hours. In all other respects the parties were under identical conditions.

On the 25th of April No. 388 developed Beri-Beri. On May 1st, No. 309 developed the disease and No. 405, 433, 347 also on May 10th, May 10th, and May 11th, respectively.

Amongst the parboiled rice party although carefully observed for possible cases of Beri-Beri no signs were noted.

If the disease is an infectious one it is difficult to explain the course of events at this place since in the one group comprising some thirty-five individuals to whom white rice was being issued there occurred five cases of Beri-Beri, while in the other group comprising some eighty-five individuals to whom parboiled rice was being issued no sign of the disease appeared. The members of the two groups occupied the same quarters, freely intermingled with one another and were so far as could be known under identical conditions in all respects save only in the matter of diet.

As regards the question of place infection the transference of the party from the 64th mile to the $61\frac{1}{2}$ mile had no effect on the progress of the outbreak and to further test the possibility of the quarters at the 64th mile being an infected place some thirty individuals who had been on parboiled rice at Durien Tipus since the 12th of October and in whom no case of Beri-Beri had occurred since that date, were removed to these quarters on May 7th. These remained in the 64th mile quarters for one month, a period exceeding by a fortnight, the duration of residence of the white rice party in these quarters before the first case of Beri-Beri occurred. Save for the change of place the conditions, dietary, etc., remained unchanged and no sign of Beri-Beri was observed among them.

As possibly further bearing upon the question of infection by one of the suggested modes, namely through faecal contamination, it may be noted that during the time the two parties were together at the $61\frac{1}{2}$ mile an outbreak of amœbic dysentery occurred. This disease showed no selective action on the parties but attacked members of both, eight cases occurring in the parboiled rice party and six cases in the white rice party.

On May 12th all the people remaining on white rice at the $61\frac{1}{2}$ mile were changed to parboiled rice. After this date no case of Beri-Beri occurred.

In view of the importance of the results in this party its history may be briefly reviewed. From October 19th to March 22nd they were

housed at Kuala Ayer Baning and were on white rice. The history of the outbreaks of Beri-Beri at Duien Tipus and Juntai had led us to believe that if the consumption of white rice were responsible for the disease, an average period of about one hundred and twenty-five days was required. This party was on white rice for one hundred and fifty-six days and no sign of the disease had appeared. It is to be remembered however, from the view point of the dietary hypothesis, that owing to the conditions which had been laid down the party became eventually one composed of selected individuals who may be presumed to have been more resistant to the possible harmful influence of white rice.

On March 23rd the party was transferred to new quarters at the 64th mile and on April 7th the first case of Beri-Beri occurred. When two definite cases had developed the party was transferred to the 61½ mile and formed part of a large party at that place. Cases of Beri-Beri continued to appear in this group until the change to parboiled rice on May 12th.

Change of place had therefore no effect upon the progress of the outbreak and no evidence was forthcoming that members of this group were able to convey the disease to individuals on a parboiled rice diet.

It is believed that these results indicate that place *per se* or considered as a nidus of infection has no influence upon the development of Beri-Beri and confirmed our belief that the disease is not a communicable one.

PARTY No. I. (*Funtai, Durian Tipus, 61½ Mile*)

October 1907—May 1908.

| | OCT. | NOV. | DEC. | JAN. | FEB. | MARCH | APRIL | MAY |
|----------|-------|------|----------------|----------------|----------------|-------|----------------|-----|
| 1 | | | | | | | | |
| 2 | | | | | Party No. III. | | | |
| 3 | | | | | Left | | | |
| 6 | | | | | | | | |
| 7 (a.s.) | Hosp. | | | | | | | |
| 8 | | | | | | | | |
| 10 | | | | | | | | |
| 11 | | | | | | | | |
| 12 | | | | | Party No. III | | | |
| 13 | | | | | | | | |
| 14 | | | | | | | | |
| 16 | | | | | | | | |
| 18 | | | | Hosp. | | | | |
| 19 | | | | Party No. III. | | | | |
| 20 | | | | | | | | |
| 21 | | | Party No. III. | | | | | |
| 23 | | | | | | | | |
| 24 | | | Party No. III. | | | | | |
| 25 | | | | | | | Party No. III. | |
| 26 | | | | | | | | |
| 28 | | | | | | | | |
| 29 | | | | | | | | |

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

30 _____
 31 _____
 32 _____

 35 _____
 36 _____
 37(B.B.) _____
 38 _____
 39 _____
 40 _____ Hosp.
 41 _____ Party No. III.
 42 _____ Died. Tuberculosis.
 43 _____

 45 _____ Party No. III. _____
 46 _____ Party No. II.

 48 _____
 49 _____
 50 _____ Party No. III.
 51 _____
 52 _____
 53 _____
 54 _____ Hospital _____ Left
 55 _____

 57(B.B.) _____ Hospital _____

 59 _____
 60(B.B.) _____ Hospital _____
 61 _____

OCT. NOV. DEC. JAN FEB. MARCH APRIL MAY

62 _____

63 _____

64 Hosp. _____ Died. Pyaemia.

66 _____

68 _____ Party No. III.

69 _____

70 _____

71 _____ Hosp. _____ Left

72 _____ "

73 _____ Party No. II.

74 _____ Party No. III.

75 _____

76 (B.B.) _____ Hosp. _____

78 _____

79 _____

80 _____ Party No. III.

81 _____ Left

82 _____

83 _____

84 _____

85 _____

86 _____

87 _____ Hosp.

88 _____

90 _____

91 _____ Left

92 _____ Hosp.

93 _____

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

| | | | |
|------------|----------|----------------|----------------|
| 95 | _____ | Hospital | _____ |
| 96 | _____ | | |
| 97 | _____ | | |
| 98 | _____ | | |
| 99 | _____ | | |
| 101 | _____ | | Left |
| 102 | _____ | | |
| 103 | _____ | | |
| 104 | _____ | | Party No. II. |
| 105 | _____ | Party No. III. | |
| 107 | _____ | | |
| 108 | _____ | | |
| 109 (B.B.) | Hosp. | _____ | Party No. III. |
| 110 | _____ | | |
| 111 | _____ | | Party No. II. |
| 112 | _____ | | Party No. III. |
| 113 | _____ | | |
| 114 | _____ | | |
| 115 | Hospital | _____ | Party No. II. |
| 116 | _____ | | |
| 117 | _____ | | Party No. III. |
| 118 | _____ | | |
| 119 | _____ | | Party No. III |
| 120 | _____ | Hospital | _____ |
| 121 | _____ | | |
| 122 | _____ | Left | |
| 123 | _____ | | |
| 124 (B.B.) | _____ | Hospital | _____ |
| 125 | _____ | | |

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

126 _____ Hospital

127(M.B.) _____ Hospital _____

129 _____ Left

131 Party No. II. _____ Party No. II. _____

132 _____

133 Java _____

134 " _____ Party No. III. _____

135 " _____ " _____

136 " _____ " _____

137 " _____ Party No. III. _____

138 " _____

139 " _____

140 " _____

141 " _____

142 " _____ Party No. II. _____

143 " _____

144 " _____

145 " _____

146 " _____ Party No. II. _____

147 Party No. III. _____

148 From Malay Kampong _____ Party No. II. _____

149 Java _____

150 " _____

151 " _____

152 " _____

153 " _____ Left

154 " _____

155 From Java. _____

156 " " _____ Hosp. _____ Hosp. _____

157 " " _____

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

| | | | | | | | |
|-----------|---------------|--------------------|----------|----------|----------|-------|-------|
| 158 | Java | _____ | _____ | _____ | _____ | _____ | _____ |
| 159 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 160 | " | _____ | Hospital | _____ | _____ | _____ | _____ |
| 161 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 162 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 163 | " | _____ | Left | _____ | _____ | _____ | _____ |
| 164 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 165 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 166 | " | _____ | _____ | _____ | Left | _____ | _____ |
| 167 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 168 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 169 | " | _____ | _____ | _____ | _____ | Hosp. | _____ |
| 170 | " | _____ | Left | _____ | _____ | _____ | _____ |
| 171 | " | _____ | Left | _____ | _____ | _____ | _____ |
| 172 | Party No. III | _____ | _____ | _____ | _____ | _____ | _____ |
| 173 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 174 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 175 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 176 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 177 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 178 | | From Party No. III | _____ | _____ | _____ | _____ | _____ |
| 179 | | " | _____ | _____ | _____ | _____ | _____ |
| 180 | | " | _____ | _____ | Hospital | _____ | _____ |
| 181 | | " | _____ | _____ | _____ | _____ | _____ |
| 182 | | Hosp. | _____ | Left | _____ | _____ | _____ |
| 183 | Party No. III | _____ | _____ | _____ | _____ | _____ | _____ |
| 184 | " | _____ | _____ | _____ | _____ | _____ | _____ |
| 185 | " | _____ | Left | _____ | _____ | _____ | _____ |
| 186 | " | _____ | _____ | _____ | _____ | Hosp. | _____ |
| 187 | | _____ | _____ | _____ | _____ | _____ | _____ |
| 188(B.B.) | | Party No. III | _____ | Hospital | _____ | _____ | _____ |

OCT. NOV. DEC. JAN. FEB. MARCH APRIL MAY

300 _____

303 Hospital Hospital

305 From Party No. II. Left

306 Hospital _____

307 " Left

314 From Party No. II. _____

316 " " _____

320 From Party No. II. Left

321 _____

325 From Hosp. Died. Tuberculosis.

338 From Party No. II. Hosp.

345 _____ Left

346 _____

352 Hospital Left

357 From Party No. II. _____

360 From Party No. II. _____

367 Party No. II. Left

369 From Party No. II. _____

370 " " _____

372 " " _____

373 _____

374 _____ Hosp.

375 Hosp. Left

376 _____ Hosp.

382 From Party No. II. _____

383 _____

387 _____

| | OCT. | NOV. | DEC. | JAN. | FEB. | MARCH | APRIL | MAY |
|-----|---------------|------|------|---------------|---------------|-------|-------|------|
| 389 | | | | | | | | |
| 393 | | | | | | | | |
| 418 | | | | Party No. II. | | | | |
| 421 | | | | | | | | |
| 422 | | | | Party No. II. | | | | |
| 434 | Party No. II. | | | | Party No. II. | | Hosp. | |
| 436 | | | | | From Hosp. | | Left | |
| 438 | | | | | | | | |
| 440 | Party No. II. | | | | | | | Left |

PARTY No. I (October 1907—May 1908).

This party, now composed of Parties No. I A. and No. I B. subsequent to the change to parboiled rice, was employed for comparison with the results in Party No. II. In the case of Party No. I A. this change dates from October 12th and in the case of Party No. I B. from October 19th. The party continued on parboiled rice until the end of the investigation in June 1908. One group remained at Durien Tipus until transferred to the 64th mile on May 7th; the other group, formerly at Juntai, was transferred to the 61½ mile on November 18th. As has already been stated no case of Beri-Beri occurred in either group composing the party subsequently to the change to parboiled rice.

On October 19th, all the cases of Beri-Beri eight in number (Nos. 7, 37, 57, 60, 76, 102, 124 and 127) were at the Durien Tipus quarters. They remained in this place until November 3rd, when seven of them were sent to Hospital at Kuala Klawang; No. 37, being then too ill to be moved, remained. Attention is called to this fact as bearing still further upon the question of infection including the influence of place. In these quarters seven cases of Beri-Beri had developed in eight weeks among an average population of about thirty. There were now eight cases of Beri-Beri in different stages in the quarters, an undoubted focus of infection if the disease were a communicable one, yet no further sign of the disease showed itself.

It may be that all the susceptible individuals in this party had by this time become affected and it may be suggested that this explains the cessation of the outbreak coincident with the change in diet. Against this stands the fact that on November 1st, fourteen labourers, who had just arrived from Java, joined this party while the Beri-Beri patients were still at Durien Tipus and on November 15th, twenty-two more such persons joined the party. It might have been expected that among these there would have been some who were susceptible.

No. 188 who had developed the disease at Pertang (Party III) on December 28th was transferred to this party on January 5th, in order to be nearer medical care. As has been already mentioned cases of Beri-Beri were also transferred to this party from Party No. II and cases of Beri-Beri continued to develop in Party No. II while occupying the same quarters as Party No. I. No attempt was made to isolate such cases, in this respect following the universal custom in the treatment of Beri-Beri. It resulted in consequence that cases of Beri-Beri in all stages of development were in contact with healthy people, yet at no time was there any evidence that the disease could be communicated to an individual on parboiled rice.

The party under review was a large one, two hundred and two individuals were present in it at one time or another from October 1907 to May 1908. One hundred and fifty-six of these were present for not less than three months continuously and one hundred and eight for not less than six months continuously.

During this period twenty-five individuals returned from hospital and twenty-one joined the party from Party No. III Pertang. From time to time also some joined from Party No. II.

Thus it will be seen that in this large party there was abundant opportunity for the introduction of an infection, yet no sign suggestive of Beri-Beri was observed in any of its members.

PARTY No. III.

This was a small party in the employ of the same contractor and engaged on his rubber estate at the village of Pertang. Only parboiled rice had been issued to the labourers on this estate from August 2nd, 1906, and there had been no cases of Beri-Beri. From August 16th, 1907, white rice was issued and continued till May 31st, 1908. The party was under observation from August 1st, 1907. As will be seen from the table the number of individuals remaining there continuously during this period was small, eight. The number on white rice continuously for at least three months was thirty-six and for at least six months there was twelve.

One case of Beri-Beri only (No. 188) occurred at this place on December 28th, 1907. He was transferred to Party No. I on January 5th, 1908.

From the point of view of infection in Party No. III the patient remained among them for seven days after he had developed the disease. Between December 28th, 1907, and January 5th, 1908, there were in all thirty people in this party. In addition it should be stated that this party was much less isolated than the other parties under observation.

As regards food their proximity to a village rendered it easy for the men to obtain food other than that supplied to them.

The white rice issued at this place was from the same stock as supplied to Parties No. I and No. II.

No special importance is attached to the results in this party, as owing to its situation, there were many uncontrolled factors in operation.

The table is given in order to make complete the history of the individuals in other parties.

PARTY No. III. (*Pertang*).

August 1907—May 1908.

| | AUG. | SEPT. | OCT. | NOV. | DEC. | JAN. | FEB. | MAR. | APRIL | MAY |
|-----|------|-------|------|------|------|------|------|------|-------|---|
| 2 | | | | | | | | | | From Party No. I. _____ |
| 12 | | | | | | | | | | _____ |
| 19 | | | | | | | | | | From Party No. I. _____ |
| 21 | | | | | | | | | | From Party No. I. _____ |
| 24 | | | | | | | | | | _____ Left |
| 25 | | | | | | | | | | From Party No. I. _____ |
| 41 | | | | | | | | | | From Party No. I. _____ |
| 45 | | | | | | | | | | From Party No. I. _____ To Party No. I. _____ |
| 50 | | | | | | | | | | _____ |
| 68 | | | | | | | | | | From Party No. I. _____ |
| 73 | | | | | | | | | | _____ |
| 74 | | | | | | | | | | From Party No. I. _____ |
| 77 | | | | | | | | | | From Party No. I. A. _____ Left |
| 80 | | | | | | | | | | From Party No. I. _____ |
| 105 | | | | | | | | | | _____ Left |
| 109 | | | | | | | | | | From Party No. I. _____ Hosp. _____ |
| 112 | | | | | | | | | | _____ |
| 117 | | | | | | | | | | _____ |
| 118 | | | | | | | | | | _____ |
| 119 | | | | | | | | | | _____ |
| 134 | | | | | | | | | | From Party No. I. _____ To Party No. I. _____ |
| 135 | | | | | | | | | | _____ |
| 136 | | | | | | | | | | _____ |
| 137 | | | | | | | | | | _____ |
| 138 | | | | | | | | | | _____ |
| 147 | | | | | | | | | | _____ To Party No. I. _____ |
| 172 | | | | | | | | | | _____ |
| 173 | | | | | | | | | | From Java. _____ |
| 174 | | | | | | | | | | _____ |

AUG. SEPT. OCT. NOV. DEC. JAN. FEB. MAR. APRIL MAY

175 From Java. _____ To Party No. I.

176 " " _____ " " "

177 " " _____ " " "

178 _____ To Party No. I.

179 _____ " " "

180 _____ " " "

181 _____ " " "

183 _____ " " "

184 _____ " " "

185 _____ " " "

186 _____ " " "

187 _____ " " "

188 _____ B.B. " " "

395 From Party No. II. _____

397 " " " _____

411 " " " _____ Left

500 _____

501 _____

502 _____

503 _____

504 _____

505 _____ Hosp. _____

506 _____

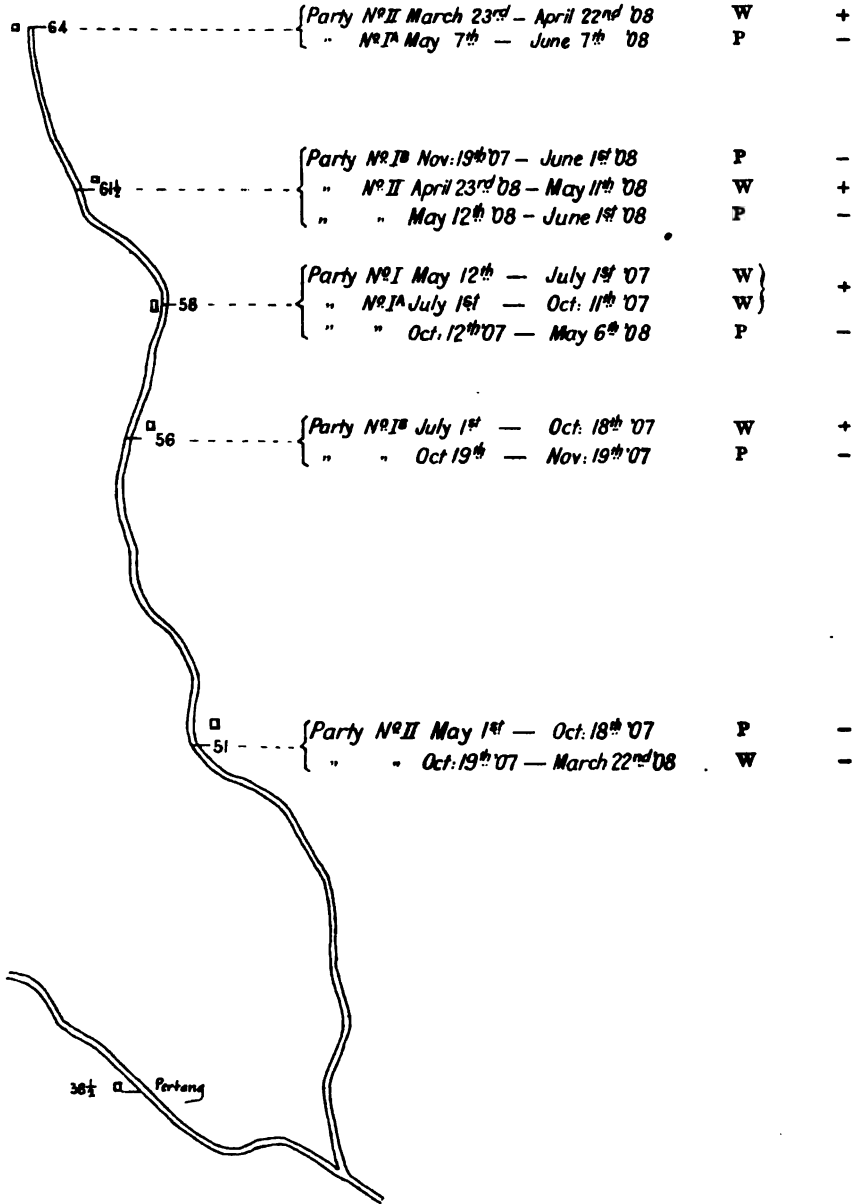
507 _____

508 _____ Left

509 _____ Left

510 _____

511 From Java. _____



Sketch showing arrangement of parties, rice consumed and results obtained.

Scale 0 2 4 Miles

W — White rice
P — Parboiled rice

Ankylostomiasis.

In view of the suggestion that has been made attributing to the presence of nematode worms in the intestine, particularly Ankylostomes, a causative action in the production of Beri-Beri, examinations were made to determine what proportion of the coolies under observation harboured these parasites. The individuals were not selected in any way so that the results below probably indicate with fair accuracy the incidence of the various intestinal parasites.

PARTY No. I.

| | | | | | | |
|---|-----|-----|-----|-----|-----|-----------|
| No ova found | ... | ... | ... | ... | ... | 26 times. |
| Ova Ankylostomes only | ... | ... | ... | ... | ... | 19 " |
| Ova Ankylostomes and Ascaris | ... | ... | ... | ... | ... | 6 " |
| Ova Ankylostomes and Tricocephalus | ... | ... | ... | ... | ... | 6 " |
| Ova Ankylostomes, Ascaris and Tricocephalus | ... | ... | ... | ... | ... | 10 " |
| Ova Ankylostomes and larvae <i>Strongyloides stercoralis</i> | ... | ... | ... | ... | ... | 1 " |
| Ova Ankylostomes, Tricocephalus and larvae <i>Strongyloides stercoralis</i> | ... | ... | ... | ... | ... | 1 " |
| Ova Ascaris only | ... | ... | ... | ... | ... | 7 " |
| Ova Ascaris and Tricocephalus | ... | ... | ... | ... | ... | 3 " |
| Ova Tricocephalus only | ... | ... | ... | ... | ... | 3 " |
| Total number of examinations | | | | | | 82 |
| Percentage harbouring ankylostomes | | | | | | 52.4% |

PARTY No. II.

| | | | | | | |
|--|-----|-----|-----|-----|-----|----------|
| Noova found | ... | ... | ... | ... | ... | 5 times. |
| Ova Ankylostomes only | ... | ... | ... | ... | ... | 18 " |
| Ova Ankylostomes and Ascaris | ... | ... | ... | ... | ... | 13 " |
| Ova Ankylostomes and Tricocephalus | ... | ... | ... | ... | ... | 5 " |
| Ova Ankylostomes, Ascaris and Tricocephalus | ... | ... | ... | ... | ... | 8 " |
| Ova Ascaris only | ... | ... | ... | ... | ... | 2 " |
| Ova Tricocephalus only | ... | ... | ... | ... | ... | 3 " |
| Ova Ascaris and Tricocephalus | ... | ... | ... | ... | ... | 4 " |
| Total number of examinations | | | | | | 58 |
| Percentage harbouring ankylostomes | | | | | | 75.8% |
| Percentage of all examinations in which Ankylostome ova were found | | | | | | 62.1% |

Examination of faeces of individuals who afterwards suffered from Beri-Beri.

| | |
|---|----------|
| No ova | 6 times. |
| Ova Ankylostomes only | 4 „ |
| Ova Ankylostomes and Ascaris | 3 „ |
| Ova Ankylostomes and Tricocephalus | 2 „ |
| Ova Ankylostomes, Ascaris and Tricocephalus | 2 „ |
| Ova Ankylostomes and larvae <i>Strongyloides</i>
<i>stercoralis</i> | 1 „ |
| Ova Ascaris and Tricocephalus | 1 „ |
| <hr/> | |
| Total number of examinations ... | 19 |
| Percentage of individuals who suffered from
Beri-Beri harbouring ankylostomes | 63.2% |

The percentage of persons harbouring ankylostomes among those who suffered from Beri-Beri was therefore practically the same as the percentage among the whole population under observation. The number of examinations is admittedly too small to be conclusive on the point, but the results indicate that Ankylostomes play no part in the causation of Beri-Beri.

Blood Examinations.

Upwards of a thousand of these examinations were made and systematic observations on the blood of the Beri-Beri patients failed to reveal, by the staining methods employed, the presence of any organism likely to have a causal relationship to the disease.

During the period of slight pyrexia which commonly preceded the onset of definite signs of Beri-Beri, malaria parasites were found in the blood on several occasions. In such cases it may be that the attack of malaria precipitated the onset of Beri-Beri or *vice versa*.

An attempt was made, by taking blood films from a number of persons, to obtain a series of films extending over some months preceding the onset of Beri-Beri in a given individual. The choice of individuals was not fortunate however and the most that was obtained was that in one case the series extended over a week preceding the onset of Beri-Beri. In this case no parasites were found.

Results and Conclusions.

1. In the course of a systematic inquiry especially undertaken to test the position of white rice as a causative agent in Beri-Beri, it was observed that twenty cases of this disease occurred among two hundred and twenty people on white rice who were continuously present in the various parties during the course of the outbreaks. In the parties on parboiled rice during the same periods and under similar conditions, among two hundred and seventy-three people no sign of the disease appeared.

2. Since all cases presenting equivocal signs of the disease were excluded we are of opinion that there were many other cases which in the ordinary routine of clinical practice would have been regarded as Beri-Beri. Such cases only occurred among people who consumed white rice, and their inclusion would not strengthen the case for an infectious origin of the disease.

3. No case of Beri-Beri occurred in any person who had been on white rice for a less period than eighty-seven days.

4. Systematic examinations were made of the blood and urine of patients suffering from Beri-Beri. Various methods of examination were employed but in no instance were any organisms found except those well known as the causative agents of other diseases.

5. In the course of the inquiry patients in various stages of Beri-Beri were at times in contact with parties of men on parboiled rice. The results of observations made on such occasions furnished evidence that the disease is not a directly communicable one.

6. Removal of patients suffering from Beri-Beri from one place to another did not influence the progress of the disease and removal of entire parties from the place where the disease had occurred did not influence the progress of the outbreak so long as they continued on white rice. These experiments suggest although they do not prove that place *per se* or considered as a nidus of infection has no influence upon the development of Beri-Beri.

7. In three instances in which definite outbreaks of Beri-Beri occurred among parties on white rice, substitution of parboiled rice was followed by a cessation of the outbreak.

8. The outbreaks of Beri-Beri cannot be attributed to deficiency in the diet issued, either in respect of proteids, fats, carbohydrates or salts.

9. No evidence was obtained to show that any article of food other than white rice was a possible source of a causative agent of the disease.

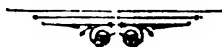
10. Ankylostomes and other Nematode worms were not found in a larger proportion of patients suffering from Beri-Beri than in the general population under observation.

11. The general results lend support to the view that the disease Beri-Beri as it occurs in this Peninsula has, if not its origin in, at least an intimate relation with white rice, and justify further research along these lines.

Samples of the rices employed in this inquiry were taken daily and submitted to exhaustive chemical analyses and microscopic observation. The results of this branch of the inquiry, which is still in progress, will be communicated in a subsequent number of these STUDIES and will form the basis for a further investigation which it is proposed to undertake.

References.

1. TAKAKI, K.: "Prevention of Kakké in Japanese Navy," *Sri-i-Kwai* 1885, 1886, 1887.
2. EIJKMAN, C.: "Polyneuritis bij Hoenders; nieuwe bijdrage tot de aetiologie der Ziekte" *Jaarverslag van Lab. v. Path. Anat. en Bakt. over het jaar 1895.*; Batavia, 1896.
3. VORDERMAN, A. G.: "Onderzoek naar het verband tusschen den aard der rijstroeding in de gevangenissen op Java en Madoera en het voorkomen van beri-beri onder de geïnterneerden," Batavia, 1897.
4. BRADDON, W. L.: "The Aetiology of Beri-beri" *Federated Malay States Medical Archives*, 1901. "The Cause and Prevention of Beri-Beri," London, 1907.
5. DUBRUEL: "Le Béribéri" Paris, 1905.
6. VAN DIEREN, E.: "Meelvergiftigingen" Amsterdam, 1907.



STUDIES

FROM

THE INSTITUTE FOR MEDICAL RESEARCH, FEDERATED MALAY STATES.

- No. 1. Vol. 1.—No. 1. The Malarial Fevers of British Malaya by HAMILTON WRIGHT, M.D. (McGILL).
- No. 2. Vol. 2.—No. 1. An Inquiry into the Etiology and Pathology of Beri-Beri by HAMILTON WRIGHT, M.D. (McGILL).
- No. 3. Vol. 2.—No. 2. On the Classification and Pathology of Beri-Beri by HAMILTON WRIGHT, M.D. (McGILL).
- No. 4. Vol. 3.—Part 1. The Diseases of British Malaya by C. W. DANIELS, M.B. CAMB.
- No. 5. Vol. 3.—Part 2. Water Supplies (Preliminary Observations) by C. W. DANIELS, M.B. CAMB.
- No. 6. Vol. 3.—Part 3. Breeding Grounds of Culicida by C. W. DANIELS, M.B. CAMB. The Culicida of Malaya by G. F. LEICESTER, M.B., C.M., I.DIN.
- No. 7. Vol. 3.—Part 4. The Outbreaks of Rinderpest in Selangor, 1903 and 1904, by C. W. DANIELS, M.B. CAMB.
- No. 8. Vol. 4.—Observations on Beri-Beri by C. W. DANIELS, M.B. CAMB.
- No. 9. Surra in the Federated Malay States by HENRY FRASER, M.D. (ABER.) and S. L. SYMONDS.
- No. 10. An Inquiry concerning the Etiology of Beri-Beri by HENRY FRASER, M.D. (ABER.) and A. T. STANTON, M.D. (TOR).

228-77

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

THE ETIOLOGY OF BERI-BERI.

BY
HENRY FRASER, M.D. (ABER.),
Director, Institute for Medical Research,
AND
A. T. STANTON, M.D. (TOR.),
Bacteriologist, Institute for Medical Research.

PRINTED BY AUTHORITY OF THE RESIDENT-GENERAL, F.M.S.

Kuala Lumpur:
PRINTED AT THE F.M.S. GOVERNMENT PRINTING OFFICE.

1939.

Digitized by Google

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

THE ETIOLOGY OF BERI-BERI.

BY
HENRY FRASER, M.D. (ABER.),
Director, Institute for Medical Research.

AND
A. T. STANTON, M.D. (TOR.),
Bacteriologist, Institute for Medical Research.

PRINTED BY AUTHORITY OF THE RESIDENT-GENERAL, F.M.S.

Kuala Lumpur:
PRINTED AT THE F.M.S. GOVERNMENT PRINTING OFFICE.

1909.

THE ETIOLOGY OF BERI-BERI.

THE suggestion of a causal relationship between the consumption of white rice and the disease beri-beri was first formally made in this country by Braddon.¹ This observer also drew attention to the important fact that those who consumed rice that had been parboiled before husking remained free from the disease, as did also the native Malays who consumed rice prepared by primitive methods of pounding and winnowing.

A series of observations made by the writers² in 1907 on two parties of labourers, under conditions which excluded or adequately controlled the operation of factors other than diet, confirmed the correctness of this view of the causation of the disease. The prior observations of Fletcher³ and Lucy⁴ in this country and of Dubruel⁵ in Indo-China and the recently published observations of Ellis⁶ furnish further testimony, and it may now be claimed that the theory rests on a solid basis of evidence.

The mechanism by which white rice was able to produce this result has remained obscure. Braddon suggested that "the cause of the disorder is not indeed rice, *qua* rice, or as an article of diet, but diseased rice; rice from which poison derived from decay, due perhaps to some fungus, or mould, or germ, or spore, originally perhaps growing upon the husk, has become mixed during the process of milling; or upon which such fungus may have grown and such poison have been produced after decortication." Eijkman⁷ from experiments on fowls concluded that a definite poison exists commonly in rice and that for this poison or its effects something in the pericarp is an antidote. Dubruel conceived the ingestion of an organism associated with white rice, which organism multiplying in the body produced the disease.

Following the line of thought suggested by the poison hypothesis researches were undertaken to determine whether from white rices actually associated with outbreaks of beri-beri there could be extracted by means of various solvents any substance or substances recognisable by chemical methods as poisonous in character. These researches

failed of their object, though it is admitted that the accuracy of the poison hypothesis was not thereby disproved.

Certain results which emerged from chemical analysis and histological examination of the rices turned attention to the possibility of an explanation of the course of events on an hypothesis of a defect of nutrition. That this explanation was inadequate, if dietary constituents as estimated by the ordinary analytical methods were alone considered, had been shown in the preliminary investigation.

By a series of experiments on domestic fowls, the details of which will be supplied in a later publication, it was shown that these animals when fed on various kinds of rice were sensitive to differences between them. By further and repeated experiments with rices known to have been associated with outbreaks of beri-beri and with controls under identical conditions fed on parboiled rice, it was established that a certain reaction in fowls might be taken as an indicator of the beri-beri-producing power of a given rice when forming the staple of the diet in man.* Whether the disease produced in fowls be accepted or not as analogous to beri-beri in man, the validity of the arguments here advanced remains unimpaired.

Rices were available that were known to have been associated with outbreaks of beri-beri, samples having been taken daily during the continuance of the preliminary enquiry in 1907. Also through the courtesy of Dr. J. D. Gimlette and Dr. G. D. Freer we were enabled to procure white rice which was being consumed prior to an outbreak of beri-beri among Malays at the Kuala Lumpur Police Depot, which outbreak ceased on changing the rice supplied to the parboiled variety. It was shown that these rices when fed to fowls constantly produced a certain disease in a large proportion of them, while parboiled rice as constantly failed to produce this result in groups under identical conditions. It is our belief that this disease, polyneuritis gallinarum, is truly analogous to beri-beri in man, similar in its etiology, in its clinical manifestations and in its pathological effects, and that its occurrence should be held as important confirmatory testimony of the connection between white rice and beri-beri. It is desirable, however, to emphasise the point that the acceptance or non-acceptance of this opinion is immaterial to the argument; for this purpose the occurrence of the disease is employed only as a reaction. The fact that certain white rices when forming the staple of a diet in man produce beri-beri rests on quite other testimony than that supplied by experiments on domestic fowls.

The commercial varieties of white rice are numerous, but in this country, apart from the grading as to quality, two varieties are in common use and are known respectively as Siam and Rangoon.

From epidemiological considerations and from experimental evidence it appears that Siam rice is considerably more potent in its beri-beri-producing powers than Rangoon rice.

The proteins, fats, carbohydrates, and ash were determined for the different varieties of rice which had been employed in the experiments, with the following percentage results calculated on dried material—

| | Protein. | | Fat. | | Carbohydrate. | | Ash. |
|-----------------------|----------|-----|------|-----|---------------|-----|------|
| White rice (Siam) ... | 9.07 | ... | 0.17 | ... | 90.11 | ... | 0.65 |
| „ (Rangoon) ... | 8.44 | ... | 0.81 | ... | 89.90 | ... | 0.85 |
| Parboiled rice... | 9.48 | ... | 0.51 | ... | 89.12 | ... | 0.89 |

A comparison of these results shows that the only marked difference among the rices was in respect of fat, which was most abundant in the variety known as Rangoon, less abundant in parboiled rice, and still less abundant in Siam rice. These observations, taken in conjunction with the experimental results in fowls, excluded the possibility of an explanation of the origin of beri-beri on the ground of a deficiency in fat. It will be noted that these analyses did not include an estimation of the relative proportions of the inorganic salts composing the ash, nor did they take account of the manner of combination, organic or inorganic, in which these substances originally existed in the rice grain.

By a method devised in this laboratory, sections of the various rice grains were obtained of sufficient thinness to permit the examination in detail of their histological characters. By suitable staining methods it was shown that in parboiled rice remnants of the pericarp remained attached to the rice grain whereas in Siam rice the pericarp and the layers subjacent to it had been polished away. It would appear that parboiling renders the grain tough and non-friable, in consequence the peripheral layers cannot be removed so readily as in the untreated grain. It was further demonstrated that the layers so retained in parboiled rice contained the most of the aleurone and oily material present in rice grains. Rice as prepared by primitive methods (Malay rice) was similarly examined, and, as might have been expected from the pounding to which this rice had been subjected,

parts of the pericarp and subjacent layers were chipped off to a varying extent, but on the whole these layers were retained to a greater extent than is the case with white rice.

Early in the course of the experiments the observation was made that parboiled rice subjected to exhaustion with hot alcohol and thereafter carefully dried in the sun to free it from alcohol, produced when fed to fowls a disease indistinguishable from that observed in fowls fed on white rice, though such parboiled rice in its original state was incapable of producing this result however long continued.

The association of the observations referred to in the two preceding paragraphs seemed to point a way to a solution of the problem. It had been shown that white rice as prepared in the mills of this country produced the same results in fowls as white rice known to have been associated with beri-beri. If now a substance or substances residing in the outer layers which are milled away in white rice and are retained in parboiled rice could be added to white rice and so prevent its harmful effects it was conceived that the nutritive hypothesis would thereby be supported.

In accordance with this idea the following experiments were initiated. A rice mill in Singapore was visited and there was obtained (A) a quantity of the original padi then being milled—in this case a partially husked padi imported from Indo-China; (B) a quantity of the finished product as it came from the machine; (C) a quantity of the “polishings” from the same rice. Polishings, it may be stated, consist of the outer layers of the seed removed in the process of making the rice white. The miller estimates that 40 parts of padi produce 25 parts of white rice, 5 parts of polishings and 10 parts of husk. The polishings are sold as food for cattle and the husks are burned as fuel in the mill.

EXPERIMENT A.—Twelve fowls were fed on the padi for five weeks.

Result. All remained healthy.

EXPERIMENT B.—Twelve fowls were fed on the white rice alone.

Result. In five weeks six had developed polyneuritis; two were dead, one having suffered from polyneuritis and one from a disease other than polyneuritis; five fowls remained healthy.

EXPERIMENT C.—Twelve fowls were fed on rice taken daily from the same bag as that used in Experiment B; in addition, polishings in the form of emulsion, in amount equal to that milled from the

quantity of rice consumed, were fed by a tube passed into the crop daily. This quantity was subsequently diminished week by week until only 3 grammes of polishings per kilogramme of body weight were being given daily. This amount sufficed to maintain the fowls in health and in constant weight.

Result. The experiment was continued for seven weeks and all remained healthy.

This result was subsequently confirmed for rice from known outbreaks of beri-beri.

It will be understood that these three experiments were in progress simultaneously and that the fowls were in all respects under identical conditions.

EXPERIMENT D.—Part of the original padi was taken and milled by a Malay woman by primitive methods into the finished product as eaten by Malays. Eight fowls, fed for five weeks on the rice prepared from the original padi by the Malay method, remained healthy. Eight fowls only were used for this experiment as the quantity of padi then remaining sufficed only for this number for the time it was estimated the experiment would last.

Attention is drawn to the important point that the products used in these experiments were all derived from the same lot of padi, and the results force us to the conclusion that it is the milling process which is essentially at fault; the polishing of white rice removes from the seed some substance or substances essential to the maintenance of the normal nutrition of nerve tissues.

To elucidate the point as to whether rice when freshly milled is less harmful than that which has become stale, an assistant was stationed in Singapore who sent daily to the laboratory by the most expeditious route a quantity of rice milled on the day of despatch. Twelve fowls were fed on this rice and five developed polyneuritis in four weeks. This result, which is similar to that obtained in other experiments, when fowls were fed on rices milled from four weeks to two years previously, disposes of the suggestion that the harmfulness of white rice is due to its staleness or to the development in it of a poisonous substance or substances subsequently to its being milled. The root of the evil lies in the milling process itself. The result further indicates the inadequacy of preventive measures founded on the poison hypothesis in regard to the use of freshly milled rice.

An experiment was now planned to determine whether a parboiled rice proved harmless could by exhaustion with hot alcohol be reduced to such a condition that it would produce polyneuritis when fed to fowls, and whether the substances so extracted when fed to fowls with a white rice proved harmful could prevent the development of polyneuritis. For this purpose parboiled rice was repeatedly exhausted with hot alcohol. The alcoholic extracts were concentrated *in vacuo* at a temperature of $52^{\circ}\text{C}.$, freed from alcohol and the residue emulsified in distilled water. Experiments with these products showed that fowls fed on the exhausted parboiled rice contracted polyneuritis, and that fowls fed on a white rice proved harmful by previous experiment remained healthy if they received in addition a quantity of the extract.

Having by these and other experiments, the details of which are omitted so as not to encumber the argument, arrived at the point when it was clear that the essential cause of beri-beri was to be sought for in a nutritive defect, further efforts were made to determine by chemical methods precise differences between various rices. Such differences, if they are to furnish an adequate explanation for the origin of beri-beri, must be in accordance with clinical observations and the experimental results in fowls.

Acting on a suggestion made to one of us by Dr. F. W. Mott, F.R.S., an attempt was made to estimate the lipoids of the different rices, but as the time element enters so largely into these estimations and our experiments with fowls were proceeding rapidly, it was decided to determine the amount of phosphorus calculated as phosphorus pentoxide (P_2O_5) in the various rices in use. It speedily became apparent from these analyses that the phosphorus content of the different rices varied with their known harmful influence, the less phosphorus contained in a given rice the more liable was it to produce polyneuritis in fowls; conversely, the higher the phosphorus content the less likely was it to produce polyneuritis.

Thus a sample of parboiled rice which was fed to fowls over many weeks all remaining healthy was found to contain .469 per cent. P_2O_5 and a sample of white rice which produced polyneuritis in fowls yielded .277 per cent. P_2O_5 . The rice polishings employed in Experiment C yielded 4.2 per cent. P_2O_5 .

From a series of observations it was determined that a fowl under the conditions of our experiments weighing from 1,200 to 1,400 grammes required 60 grammes of parboiled rice daily to maintain it in

health and in nutritive equilibrium. In Experiment C it was determined experimentally, the chemical analysis being then unknown, that when fed on white rice a fowl of this weight required the addition of about 8.5 grammes of polishings to preserve it in nutritive equilibrium. From the data given above it may readily be calculated what amount of polishings added to white rice is required to raise the phosphorus content of the white rice diet to that of the parboiled rice diet. Thus

| | |
|------------------------------------|--------------------------|
| 60 grammes of parboiled rice ... | 3,120 grms. P_2O_5 , |
| 60 " white " ... | <u>1,662 "</u> |
| Difference ... | 1,458 " P_2O_5 . |

Polishings contain 4.2 per cent. phosphorus pentoxide.

Calculated from the phosphorus content therefore 3.47 grammes of polishings added to the 60 grammes of white rice supplied to a fowl of 1,200-1,400 grammes weight should preserve it in nutritive equilibrium. From experimental observation 3.5 grammes of polishings had been shown to accomplish this result.

There is thus afforded striking testimony to the value of phosphorus estimation as an indicator of the likelihood of a given rice to produce polyneuritis when fed to fowls, or, following the argument, the likelihood of its producing beri-beri when forming the staple in the diet of man.

We are greatly indebted to Mr. B. J. Eaton, Chemist in this Institute, for valuable assistance in the chemical part of this investigation.

Dr. H. Schaumann has had the goodness to send us recently a copy of his address before the German Society of Tropical Medicine in which he stated his conjecture (*Vermutung*) that beri-beri was a disease of metabolism associated with a diet deficient in organically combined phosphorus.

SUMMARY.

1. Beri-beri is a disorder of nutrition and, as it occurs in this country, is associated with a diet in which white rice is the principal constituent.

2. White rice as produced in the mills here commonly makes default in respect of some substance or substances essential for the maintenance of the normal nutrition of nervous tissues. These substances exist in adequate amount in the original grain and in superabundant amount in the polishings from white rice.

3. The estimation in terms of phosphorus pentoxide of the total phosphorus present in a given rice may be used as an indicator of the beri-beri-producing power of such rice when forming the staple of a diet in man.

The prevention of beri-beri in this country will be achieved by substituting for the ordinary white rice a rice in which the polishing process has been omitted or carried out to a minimal extent, or by the addition to a white rice diet of articles rich in those substances in which such white rice now makes default. One such article which is cheap and may readily be obtained is the polishings from white rice.

The use of parboiled rice as suggested by Dr. Braddon will achieve a like result, provided that the polishing process is not carried beyond the limited extent now customary.

The details of the experiments upon which these conclusions are based will be supplied in a forthcoming number of these Studies, which will also include the results of further experiments now in progress.

REFERENCES.

1. Braddon, W. L.—“The Etiology of Beri-beri,” *Federated Malay States Medical Archives*, 1901. “The Cause and Prevention of Beri-beri,” 1907.
2. Fraser, H., and Stanton, A. T.—“An Inquiry Concerning the Etiology of Beri-beri,” *Lancet*, 1909; “An Inquiry Concerning the Etiology of Beri-beri,” *Studies from the Institute for Medical Research*, 1909.
3. Fletcher, W.—“Rice and Beri-beri,” *Lancet*, 1907; “Rice and Beri-beri,” *Journal of Tropical Medicine and Hygiene*, 1909.
4. Lucy, S. H. R.—“Address, British Medical Association, Penang, 1905.”
5. Dubruel—“Le Béri-béri,” 1905.
6. Ellis, W. G.—“Uncured Rice as a Cause of Beri-beri,” *British Medical Journal*, 1909.
7. Eijkman, C.—“Polyneuritis bij Hoenders,” *Jaarverslag van Lab. v. Path. Anat. en Bakt.*, Batavia, 1896.

PRINTED AT THE
F.M.S. GOVERNMENT PRESS,
KUALA LUMPUR.

STUDIES

FROM

THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

No. 12.

THE ETIOLOGY
OF
BERI-BERI

BY

HENRY FRASER, M.D. (ABER.),

Director, Institute for Medical Research,

AND

A. T. STANTON, M.D. (TOR.),

Bacteriologist, Institute for Medical Research.

Singapore:

KELLY & WALSH, LIMITED, PRINTERS,

AND AT

SHANGHAI, HONGKONG AND YOKOHAMA.

1911.

Price: Seven Shillings.

THE ETIOLOGY OF BERI-BERI.

THE ETIOLOGY OF BERI-BERI.

STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

No. 12.

THE ETIOLOGY
OF
BERI-BERI

BY
HENRY FRASER, M.D. (ABER.),
Director, Institute for Medical Research.

AND
A. T. STANTON, M.D., (TOR.)
Bacteriologist, Institute for Medical Research.

Singapore:
KELLY & WALSH, LIMITED, PRINTERS,
AND AT
SHANGHAI, HONGKONG AND YOKOHAMA.

1911.

Price: Seven Shillings.

P R E F A C E.

The inquiry, the results of which are submitted in the accompanying report, was begun under instructions from the Government of the Federated Malay States and we take this opportunity of recording our appreciation of the support extended by His Excellency the High Commissioner and other officials in furthering the interests of the research.

To Dr. G. Grijns of Weltevreden, Java, and to Dr. W. L. Braddon of Seremban, whose masterly researches on the etiology of Beri-beri formed the foundations on which we have attempted to build, as well as to many other fellow workers we are indebted for helpful criticism. For valuable assistance at various stages of the inquiry we have to thank our colleagues Dr. W. Fletcher, Mr. B. J. Eaton and Mr. J. R. Hill.

H. F.

A. T. S.

CONTENTS.

| | PAGE. |
|---|-------|
| Introduction - - - - - | I |
| Rice—histological and chemical - - - - - | 17 |
| Feeding experiments on animals - - - - - | 28 |
| The Poison hypothesis - - - - - | 34 |
| Experiments with various white rices - - - - - | 40 |
| Starvation and forced feeding experiments - - - - - | 43 |
| Experiments with unpolished rices - - - - - | 45 |
| Experiments with rice meal or polishings - - - - - | 49 |
| The Phosphorus content of rices - - - - - | 51 |
| The effect on protective substances of exposure to high
temperatures - - - - - | 63 |
| Experiments to determine the nature of the protective
substances - - - - - | 66 |
| Conclusions - - - - - | 85 |
| References - - - - - | 88 |

THE ETIOLOGY OF BERI-BERI.

THE etiology of beri-beri has been the subject of investigation by workers in many countries, and in view of the wide divergence of the conclusions arrived at and the varying results of the preventive measures suggested, it will be apparent that the problem is one of great complexity. Not a little of this confusion has been introduced by the lack of agreement among authorities as to what disease conditions are to be included under the name beri-beri. A survey of the voluminous literature which concerns itself with this disease will show not only that forms of polyneuritis of different etiology have been called beri-beri, but that even diseases of which polyneuritis may not be a prominent feature such as "epidemic dropsy" "ship beri-beri" "Ceylon beri-beri" and the like have been included under this name. It is not to be expected therefore that any single etiological factor will satisfactorily explain all the recorded outbreaks of so-called beri-beri.

The Malay Peninsula has long been known as an "endemic focus" of beri-beri and the recorded sickness and mortality rates from that disease during the past several decades show that great economic losses have resulted from its ravages. The Government of the Federated Malay States have in consequence greatly interested themselves in furthering investigation into the cause of beri-beri and five years ago the task was assigned to us of attempting to define more narrowly the etiological factors concerned. In previous papers the results of this work as obtained from time to time have been set forth and it is now proposed to review the progress made and to submit the details of the work upon which our conclusions are based.

At the time this inquiry was begun, students of the disease were divided into two principal groups in regard to their views of its origin. On the one hand it was held that beri-beri had its origin in some organism, bacterial or protozoal, and that the disease was communicable, directly or indirectly, from man to man (Manson, Scheube, Daniels, Wright); on the other hand it was maintained, more particularly by physicians in Eastern Asia where the disease is most common, that the cause of beri-beri was to be sought for in food.

An examination of the evidence put forward by these latter workers, notably Takaki in Japan, Eijkman, Grijns, Vorderman and others in the Dutch East Indies and Braddon in this country made it difficult not to believe that the question of diet was a factor of great importance in the causation of beri-beri and that inquiry along this line held out the best prospect of success.

Attention was directed long ago to the influence of diet in the causation of beri-beri. Wernich (1) who studied the disease in Japan writing in 1878 says: "The kak-ké is a chronic constitutional disorder of blood-making and of the vascular system. Rice as the exclusive food of the people is answerable for it in a quite especial way. Not, however, as some have thought because it is used in a decomposed state, but because it is used in such quantities that the power of assimilation is gradually lost for other kinds of food; and even the large quantity of rice is unable to render the nutrition and blood-making adequate."

Van Leent (2) from his experiences in the Dutch East Indies wrote: "The dietetic error which I regard as the one and only cause of the morbid composition of the blood in beri-beri consists in the too small proportion of albuminous substances and fat." In support of this he gives the experiences of the disease in the Dutch East Indian fleet from 1870 to 1878 particularly during the war in Acheen both among the native and European members of the crews.

Takaki (3) believed the disease to be due essentially to nitrogen starvation and in 1884 introduced certain reforms in the diet of the Japanese Navy. Up to this time beri-beri had been an important source of invaliding and mortality among the sailors, of whom about 30% were incapacitated annually from that disease. By the beginning of 1890 under the improved diet beri-beri was wholly eradicated and the incidence of other diseases greatly decreased. Takaki still adheres to this view of beri-beri causation and in the course of a series of lectures on "The Health of the Japanese Navy and Army" in 1906 (4) he gives fresh evidence in its support: "Another illustration of the influence of diet upon the health of the men is shown by the fact that there was not one case of beri-beri among the sailors of the naval brigade before Port Arthur although there was a large number of cases in the army. These men lived among the soldiers and under exactly the same conditions but they differed from the soldiers in one respect that they were supplied with one pound of meat, ten ounces of barley, and twenty ounces of rice *per diem* while the soldiers were supplied with five ounces of meat and thirty ounces of rice. The above example confirms my view that beri-beri largely occurs among men who are fed with an insufficient quantity of nitrogenous food and and excess of carbohydrates."

Durham (5) who studied the disease on Christmas Island and in the Malay Peninsula while concluding that: "so far as there was any semblance of a positive result in the observations it is suggestive that beri-beri is communicated from person to person more or less directly or through fomites as an actual infection" stated that "It was difficult not to believe that the presence of some constituent of the diet had a sheltering effect on the nervous system of the individual" and again that "it is suggested that certain articles of diet by virtue especially of containing phosphorized and fatty matters may tend to ward off the disease when given in sufficient amount."

Professor Chittenden quoted by Bryce ("British Medical Journal" Dec. 11, 1909) says with regard to the statement, that

the Japanese Navy had found that a high protein diet was accompanied by a diminution in the number of cases of beri-beri he contended that it did not necessarily follow that the increase of protein was the cause. He thought that it was much more likely that other elements were introduced into the food capable of accounting for the disappearance of the disease. Protein pure and simple is unlikely to be utilised as a food in the body. It is much more likely that it requires to become a salt of lime, potash or soda before it can be available for dietetic purposes. He thinks the mineral salts introduced with the protein prevented beri-beri much in the same way that lemon juice prevents the appearance of scurvy.

Epidemically and endemically the occurrence of beri-beri is certainly greatest in those tropical and subtropical countries whose inhabitants partake of a diet in which rice forms an important and generally the staple constituent. The other articles of diet vary with the country but rice varies merely in kind or quality and but little in regard to the quantity consumed.

In those countries where the disease is endemic the incidence has always been greatest among the poorer classes, inmates of public institutions and the like. Supporters of dietary hypotheses have therefore sought to account for the occurrence of the disease in that the diet consumed was deficient in some substance or substances essential for nutrition and those workers who have incriminated rice consider either that a diet in which this article bulks largely is deficient in protein or fat, or that the rice has become diseased from the action of moulds or other organisms.

The suggestion of a relationship between a rice diet and beri-beri is a very old one and there are numerous references to it in the literature of the disease. In the second edition of Hirsch's "Handbook of Geographical and Historical Pathology," published in 1881, a review is given of the various hypotheses propounded to explain the origin of beri-beri:

Dr. Hirsch says: "The conjecture that the morbid poison is some *specifically noxious thing in the food*, brought about by the local conditions, is a probable one, and there has been no lack of hypotheses in that sense. At an early period the question was propounded whether it might not be some *poisonous property of (decomposed) rice* that represented the cause of the malady."

This conjecture was revived by Braddon (6) who studied the disease in the Federated Malay States and the contributions made by this observer have led to material advances in our knowledge. Braddon was the first to show the closeness of the relationship which exists in this country between the consumption of white rice as a staple article of diet and the disease beri-beri. Of even greater importance was his demonstration of the fact that where the staple article of diet is rice which had been parboiled before husking as used by the Tamil population, or rice as prepared by the Kampong Malays, the disease does not occur.

As will presently appear this position which for many years Braddon defended in local medical circles has since been abundantly justified by controlled experiments.

Braddon's conception of the mechanism of beri-beri causation through white rice ("stale uncured rice") was that "the cause of the disorder is not indeed rice, *quâ* rice, or as an article of diet, but diseased rice; rice from which some poison derived from decay, due perhaps to some fungus, or mould, or germ, or spore originally perhaps growing upon the husk, has become mixed during the process of milling; or upon which such fungus may have grown and such poison have been produced after milling."

In accordance with this hypothesis Braddon recommended as preventive measures the use of fresh rice (Malay rice or freshly milled rice), or rice that had been parboiled before

husking. He conjectures that in the parboiling process, the hypothetical organisms are made to germinate and in that vulnerable stage are destroyed by heating. He compares the process to that employed in laboratories to sterilize or render free of noxious germs various materials which may contain them.

This hypothesis of beri-beri causation has not met with acceptance by the later investigators, Grijus, Kiewit de Jonge, Schaumann, Aron and others, who regard the disease as the result of some defect in the composition of the foodstuffs ingested.

With a view to determine the position of white rice in regard to the causation of beri-beri, Dr. Fletcher (7) carried out a series of observations extending over two years among the patients in the Lunatic Asylum, Kuala Lumpur. In his report of the results Dr. Fletcher says: "During the year 1905 a large number of lunatics in the Kuala Lumpur Lunatic Asylum suffered from beri-beri. Ninety-four of the two hundred and nineteen lunatics treated in the Asylum were affected and twenty-seven died from the disease.

"With the purpose of testing Dr. BRADDON'S theory, observations were carried on in the following years (1906, 1907) with regard to the diet of the patients. Half of the patients were placed on a diet of 'cured' (parboiled) rice while the other half remained on the diet of 'uncured' (white polished) rice which kind all the lunatics of the Asylum had been eating previous to the commencement of the observations."

"The lunatics were housed in two exactly similar buildings on opposite sides of a quadrangle surrounded by a high wall. On December 5, 1905, all the lunatics at that time in the hospital were drawn up in the dining shed and numbered off from the left. The odd numbers were subsequently domiciled in the ward on the east side of the courtyard and no alteration was made in the diet; they were still supplied with the same uncured rice as in 1905. The even numbers were quartered in the ward on the west side of the quadrangle and received the same rations as the occupants of the other ward, with the exception that they were supplied with 'cured' rice instead of the 'uncured' variety. The two batches of patients were kept in separate wards and fed at different times. Separate cooking and feeding utensils were used but otherwise the patients were allowed to associate together. At the half year the two batches were changed over to each others apartments."

The result for 1906 was that "out of 124 inmates fed on 'uncured' rice 34 suffered from beri-beri, 2 of whom were

suffering from the disease on admission and 18 died; whereas among 123 inmates fed on cured rice there were only 2 cases of beri-beri both of whom were suffering from the disease on admission, and no deaths."

At the end of 1906 there remained 35 lunatics in the "cured" rice ward and 30 in the "uncured" rice ward. The first patient admitted in 1907 was admitted to "uncured" rice, the second to "cured," the third to uncured and so on to the end of the year.

During the year 1907, 136 patients were treated in the "uncured" rice ward; of these patients 28 suffered from beri-beri, 4 of whom were suffering from the disease on their admission. During the same year 131 patients received a diet containing "cured" rice; four of them were admitted actually suffering from beri-beri but none of these 131 patients developed the disease in the Asylum.

Fletcher concludes that: "The cause of beri-beri is to be sought for in the diet. The result of the experiment tends to show that white polished rice, although of the best quality, is a cause of beri-beri acting either by some poison which it contains or by a starvation due to some defect in the nutritive value of such rice."

— — — — —

In 1907-1908 the writers carried out a series of observations designed to test the position of white rice as a causative agent in the production of beri-beri. A detailed account (8) of this inquiry has already been published and the results may here be summarized.

It was considered important that these observations should be made in a place hitherto free from the disease and where the operation of factors other than diet could be excluded or adequately controlled. Satisfactory conditions were obtained at Durien Tipus in Negri Sembilan where some three hundred Javanese labourers were engaged in the construction of a new road through virgin jungle, remote from the complex conditions which interfere with observations in populous areas. The quarters occupied by these labourers were new and the sanitary conditions were satisfactory.

The three hundred labourers were divided into two parties of approximately equal numbers and were housed some miles apart. Before beginning the experiment an examination was made of each person and the presence of cases of existing or recent beri-beri was thereby excluded.

To one party white rice (No. 2 Siam) was issued as the staple article of diet, and to the other party parboiled rice. In about three months cases of beri-beri began to occur among the members of the party on white rice. When a certain number of cases had been noted white rice was discontinued and thereafter no cases occurred. No sign of the disease appeared among the control party on parboiled rice.

The conditions were then reversed. The party hitherto on parboiled rice were given white rice and after a somewhat longer interval than in the first instance, beri-beri broke out in this group also. This outbreak ceased on discontinuing the issue of white rice. Again no sign of the disease appeared among

the control party on parboiled rice. By the transfer of individuals suffering from beri-beri and of whole groups in which the disease was occurring it was found possible to test the influence of place considered as a nidus of infection and also to test the possibility that the disease was communicable from one individual to another.

The average daily ration was as follows :—

| | | | | | | |
|-----------------|-----|-----|------|-----|-----|---------|
| Rice | ... | ... | 21.3 | oz. | 603 | grammes |
| Dried Salt Fish | ... | ... | 4.25 | " | 120 | " |
| Onions | ... | ... | 1.75 | " | 50 | " |
| Potatoes | ... | ... | 1.75 | " | 50 | " |
| Coconut oil | ... | ... | 0.85 | " | 24 | " |
| Coconut | ... | ... | 1.50 | " | 42 | " |
| Tea | ... | ... | 0.12 | " | 3.4 | " |
| Salt | ... | ... | 0.1 | " | 2.8 | " |

In a recent publication Dr. H. Schaumann (9) has dealt with the composition of this diet and quotes the results in support of his view that the disease beri-beri is due to a diet defective in substances having a high organically-combined phosphorus content.

As Dr. Schaumann has assigned values to the various food-stuffs comprising this diet which differ from those given by us it would appear that he had not before him a copy of the publication in which these were furnished, namely No. 10 of this series published in 1909. Also as the authorship of the paper published in "The Lancet" (1909 No. 4459 page 451) is wrongly quoted throughout the text it may be assumed that Dr. Schaumann had not an opportunity of consulting it in the original. In these circumstances we venture to direct attention to some errors into which we believe Dr. Schaumann has fallen.

The analyses upon which our calculations were based were carried out with the actual foodstuffs employed, and were not taken upon the authority of a text book on dietetics. We feel called upon to make this explanation as a protest against the use

of analyses of foodstuffs given in text books, however eminent the authority, for the calculation of the relative values of diets. As we shall hope to show later, fundamental differences may exist in the nutritive value of various combinations of foodstuffs which are not revealed by the ordinary methods of analysis and there are factors in the composition of food which are of much more complex nature than its protein, fat and carbohydrate content or its calorific value.

Observations have shown that beri-beri is definitely associated with the continuous consumption of a diet of which white rice is the staple and must be due either to a poison contained in white rice or to a deficiency in such a diet of some element of high physiological value. If the latter, then accuracy is imperative and can only be attained by analysis of the actual foodstuffs eaten by those among whom the disease occurred. So far as we are aware such an opportunity has only been afforded by our planned observations extending over twelve months and which are now under discussion.

The various articles composing the diet issued were submitted to analysis in this Institute by Mr. B. J. Eaton.

The average of a considerable number of analyses was as follows:—

| | Moisture. | Protein. | Fat. | Carbohydrate. | Ash. |
|---------------------|-----------|----------|------|---------------|-------|
| | % | % | % | % | % |
| White rice ... | 13.85 | 7.45 | 0.17 | 78.02 | 0.51 |
| Parboiled rice ... | 14.03 | 7.8 | 0.53 | 76.92 | 0.72 |
| Dried salt fish ... | 48.1 | 35.7 | 2.96 | | 13.24 |
| Onions ... | 86.44 | 1.5 | 0.28 | 11.28 | 0.5 |
| Potatoes ... | 65.3 | 1.8 | 0.2 | 31.66 | 1.04 |
| Coconut ... | 45.3 | 3.95 | 34.6 | 15.2 | 0.95 |

Based on these analyses the diet issued to those on white rice as the staple was calculated to consist of:—

| Protein. | Fats. | Carbohydrate. | Salts. |
|---------------|---------------|----------------|----------------|
| 91.45 grammes | 43.70 grammes | 499.16 grammes | 23.06 grammes. |

The diet issued to those on parboiled rice was calculated to consist of:—

| Protein. | Fats. | Carbohydrate. | Salts. |
|---------------|---------------|----------------|----------------|
| 93.56 grammes | 45.88 grammes | 492.54 grammes | 24.33 grammes. |

Dealing with this diet Dr. Schaumann, on page 79 of his monograph, gives a table which shows, assuming white rice to contain 7.8 % Protein, that the diet received by those on white rice contained 103.7 grammes of Protein, whereas calculated from the analyses of the actual foodstuffs supplied this diet contained 91.45 grammes of Protein. Again on page 81, assuming parboiled rice to contain 9.05 % Protein he states that the diet of those on parboiled rice contained 111 grammes of Protein or 7.3 grammes *more* than that contained in the white rice diet whereas the fact is that the parboiled rice diet contained 93.56 grammes of Protein or 2.1 grammes more than that contained in the white rice diet.

Again on page 339 Dr. Schaumann compares the diet of those on parboiled rice with that of those on white rice. The analysis of the foodstuffs there given differ materially from the analysis of the actual foodstuffs issued. By his calculations Dr. Schaumann now shows that those consuming the parboiled rice diet receive *less* protein than those consuming the white rice diet, as he here takes it that parboiled rice contains less Protein than white rice (6.38 % Protein for parboiled rice and 7.03 % Protein for white rice) which is not in accord with the statement made previously by him.

On page 363, Table XII, Dr. Schaumann gives the composition of the various foodstuffs in respect of Protein, Fat, Carbohydrate, Fibre, Total Ash and Phosphorus Pentoxide, these he takes as the basis for his final calculations.

It is possible that a parboiled rice may be found which yields 2.08 % of fat and 3.57 % of ash but among the numerous specimens examined in this Institute we have not met with such a rice.

Whatever methods of analysis are employed it may well be that the defect of nutrition, which we regard as the cause of beri-beri, will escape observation but certainly it appears that when the actual foodstuffs issued in a diet are not the subject of analysis, the results are not likely to help materially towards a solution of the problem of beri-beri causation. The valuable work which Dr. Schaumann has accomplished in this domain of research would in our opinion have been greatly enhanced in value had the opportunity been available to him of studying the actual dietary conditions under which beri-beri arises.

It is generally accepted that the dietary requirements per kilogramme of body weight are Protein 2 grammes, Fat 1.5 grammes, Carbohydrate 6 grammes, Salt 0.5 gramme. The average body weight of the people under our observation was 100 lbs. nearly—the diet should therefore contain—

| Protein. | Fat. | Carbohydrate. | Salt. |
|------------|------------|---------------|------------|
| 90 grammes | 68 grammes | 272 grammes | 20 grammes |

Comparison of this calculated standard diet with the diets consumed by the persons under observation will show that the issued diets cannot be regarded as deficient save in respect of fats. This latter deficiency is however more than compensated for by the excess of carbohydrates. With a sufficiency of protein in a diet, fats and carbohydrates are to a great extent interchangeable and the inhabitants of warm countries habitually consume less fat and more carbohydrate than do the inhabitants of cold countries.

It will be noted that the analyses of these foodstuffs did not include an estimation of the relative proportions of the inorganic salts composing the ash, nor did they take account of the manner of combination organic or inorganic, in which these substances originally existed in the rice grain. This matter will be referred to in some detail at a later stage of this report.

The conclusions arrived at as a result of this inquiry were stated as follows:—

1. In the course of a systematic inquiry especially undertaken to test the position of white rice as a causative

agent in beri-beri it was observed that twenty cases of this disease occurred among two hundred and twenty people on white rice who were continuously present in the various parties during the course of the outbreaks. In the parties on parboiled rice during the same periods and under similar conditions, among two hundred and seventy-three people no sign of the disease appeared.

2. Since all cases presenting doubtful signs of the disease were excluded we are of opinion that there were many other cases which in the ordinary routine of clinical practice would have been regarded as beri-beri. Such cases only occurred among people who consumed white rice and their inclusion would not strengthen the case for an infectious origin of the disease.

3. No case of beri-beri occurred in any person who had been on white rice for less than eighty-seven days.

4. Systematic examinations were made of the blood and urine of patients suffering from beri-beri. Various methods of examination were employed but in no case were organisms found other than those well-known as the causative agents of other diseases.

5. In the course of the inquiry patients in various stages of beri-beri were in contact with parties of men on parboiled rice. The results of observations made on such occasions furnished evidence that the disease is not a communicable one.

6. Removal of patients from the place where they had contracted the beri-beri did not influence the progress of the disease and the removal of entire parties from the place where the disease had occurred did not influence the progress of an outbreak so long as they continued on white rice.

These observations suggest that place *per se* or considered as a nidus of infection has no influence upon the development of beri-beri.

7. In three instances in which definite outbreaks of beri-beri occurred among parties of men on white rice, substitution of parboiled rice was followed by cessation of the outbreak.

8. The occurrence of beri-beri cannot be attributed to deficiency in the diet issued in respect of either protein, fat, carbohydrate or salts as estimated by the methods in common use.

9. No evidence was obtained to show that any article of the diet other than white rice was responsible for the occurrence of beri-beri.

10. Ankylostomes and other nematode worms were not found in a larger proportion of patients suffering from beri-beri than in the general population under observation.

11. The general results support the view that the disease beri-beri as it occurs in the Malay Peninsula has an intimate relationship with the consumption of white rice and further research along these lines is justified.

On the 26th of April 1909, Dr. J. D. Gimlette reported to the Senior Medical Officer, Selangor that a number of Malays recently enlisted into the Police Force and then stationed at the depôt Kuala Lumpur had reported themselves sick. On examination Dr. Gimlette had found that they were suffering from beri-beri. He adds "The occurrence is of interest because it has been possible to recognise the disease in an early stage. The majority of those attacked are newcomers who have recently fallen ill

while their rice (No. 2 Siam) has, I understand, only recently been supplied through a new sub-contractor. The patients are all Malays with one exception, a Javanese."

The rice was changed from white polished rice to parboiled rice on April 27th, and on June 2nd, Dr. Gimlette reported that the occurrence of the disease had ceased and that no deaths had occurred among those suffering from beri-beri.

Throughout the course of the inquiry at Durien Tipus samples of the white rice issued were collected daily and forwarded to the Institute.

Through the courtesy of Dr. Gimlette and Dr. Freer, Senior Medical Officer Selangor, we were enabled to obtain a bag of the white rice which was in use at the depôt at the time of the outbreak of beri-beri.

So far as the laboratory aspect of the question was concerned therefore an abundant supply of the rices which actually caused these outbreaks of beri-beri was secured. In connexion with the plan of work which we had formed we regard this circumstance as of the first importance. It could have led to no real advance in knowledge if analyses of a series of rices from various sources had been carried out without regard to their connexion with outbreaks of beri-beri. Having first shown that the consumption of a certain white rice was the cause of an outbreak of beri-beri we were in a position to proceed further and by analysis of and experiments with that rice to seek for the explanation of this relationship. The continuity of work thereby established we believe to be one of the principal merits of the observations here recorded.

Rice.General.

The cultivation of rice has existed from ancient times. It is a cereal indigenous to certain areas in both hemispheres but its culture now extends over wide areas in tropical and subtropical countries possessing an assured and heavy rainfall; also with the aid of irrigation, cultivation has been extended to areas not possessed of a sufficient rainfall.

The operation of these factors has produced many species and varieties of rice so that their number is now very great. We are not in a position to treat this subject in a comprehensive way and must of necessity confine our remarks to the countries and their rices which have come under our immediate observation.

In the Straits Settlements and Federated Malay States a limited amount of rice is grown. The quantity is quite inadequate to supply the demand of a country which is rapidly and extensively being opened up and therefore supplies have to be imported from the great producing countries more especially Siam, Burma and French Indo-China.

Rice is derived from the fruit, botanically a caryopsis, of plants belonging to the genus *Oryza*.

The product as it reaches this country is in the form of unhusked fruit, the partially husked fruit, or the finished product rice.

The imported unhusked or partially husked grain is converted into rice in the mills of Penang and Singapore. So far as can be ascertained the principal reason for the importation of the unfinished product is in order that parboiled rice may be prepared locally and sold at a lower price than the parboiled rice imported from India. The local mills are of course adapted to the production of ordinary white rice and the question as to which variety of rice a mill shall produce is answered by the demand.

The indigenous natives (Malays) in the country districts grow the grain in quantities sufficient for their own requirements and from it prepare the rice by primitive methods of pounding and winnowing.

The oriental immigrants, natives of India, China and Java, come to this country either as workers on mines or on estates and therefore have not the opportunities for growing the rice they require. The immigrant Indians are for the most part recruited from the south of India and prefer parboiled rice, which must be cheap. It is to meet this demand that the large rice mills of Penang, Singapore and Perak have been called into existence. The natives of China and Java prefer a rice which has not been parboiled and for their requirements the grain is husked and polished by machinery. The product is commercially known as Rangoon, Siam or as we prefer to call it here white rice.

The kinds or forms of rice used in this country may therefore for purposes of this account be grouped under three divisions Parboiled rice, Native or Malay rice and white rice.

Parboiled Rice.

As prepared in the large power-mills the grain is soaked in water for 24—48 hours, the water is then run off and the grain is transferred to cylinders which are lightly covered and steam is passed through the contents for five or ten minutes. The grain is thereafter transferred to open paved courts and dried in the sun. The husk is now more readily detached than in the untreated grain but the contents of the grain have been rendered tough and semi-translucent.

In the milling of this parboiled grain the husk is removed and the rice is subjected to a limited amount of pearling or polishing in a machine provided with stone facings. By no process can such rice be made to appear white, consequently polishing is as a rule employed merely to complete the removal of fragments of husk and most of the pericarp.

Parboiled rice is also prepared on a less pretentious scale in small mills not provided with steam plant. The product is similar to that produced in the larger mills but in view of the insanitary conditions prevailing in many of these places, numbers of them have been compulsorily closed.

Parboiled rice prepared in this manner has a peculiar disagreeable penetrating odour caused by the preliminary soaking in cold water; by soaking the grain in hot water in place of cold water and for a shorter period, the occurrence of this disagreeable odour can be prevented.

Other manufacturers have sought to improve their product by subjecting the grain to more extensive polishing or pearling; as will be shown later this may be attended with dangerous results to those who consume such a highly-polished parboiled rice as the staple of their diet.

According to Hooper (10) rice in Bengal is treated before husking by three methods—

- (1) Hot water is sprinkled over the padi.
- (2) The padi is soaked in cold water for 24 hours.
- (3) The padi is first soaked in water and afterwards it is boiled.

In each case the padi is thereafter dried in the sun or by other means. When the grains are sufficiently dry they are husked in a pestle and mortar.

Watt (11) states that "In India a large part of the rice sold in shops and exported to Europe as an article of food has been prepared by being first half boiled then dried in the sun and finally husked by the ordinary pestle and mortar. Such rice is in trade termed "parboiled." Husking without boiling is a very tedious process when done by hand."

According to the same authority, in India proper power mills for the preparation of rice are very few in number. In 1904 there

were in all India 127 rice mills with 17,814 employees and of this number 114 mills with 17,016 employees were located in Burma.

Parboiled rice prepared in India has no objectionable odour. It is imported into this country to a limited extent for consumption by the more affluent natives of India.

The objectionable odour possessed by the locally prepared parboiled rice has hitherto militated against the more widespread use of this kind of rice by others than natives of India and if the consumption of parboiled rice is to be encouraged such improvements in the process are required as will produce a rice similar to the parboiled product of India.

White rice.

In the milling of white rice in large power mills the padi is first deprived of the husk. This is done by passing it into a machine called the "huller" which consists of two iron discs faced with a cement of which emery is one of the ingredients. The padi enters in a stream at the centre and is driven by centrifugal action between the discs to the periphery. By this means the husk is cracked. The mixed rice and husk are now passed over the winnowing fans which blow away the husk.

The grain still covered with its brown, yellowish or other coloured pericarp now passes to a machine in which the whitening process takes place. This machine consists of a conical drum revolving at a high velocity; the drum is faced with emery cement and is surrounded by a casing lined with steel wirecloth. In the more modern mills the space between the cone and the wirecloth through which the rice passes can be altered by vertical adjustment of the spindle so that the necessary amount of attrition of the rice is secured and the desired amount of milling attained. In this process the fruit wall or pericarp, the layers subjacent to it (the subpericarpal layers) as well as the embryo are removed.

The whitened grain now passes to the polisher which consists of a revolving conical drum covered with strips of sheepskin, the whole being surrounded by a casing lined with wire-cloth. The polishing is accomplished by the rice passing down between the sheepskins and wirecloth.

In the milling and polishing machines the rice meal or "polishings" passes through the wire-cloth casings and is collected. Rice polishings are commonly used as a foodstuff for cattle and pigs. We are informed that the natives in the neighbourhood of the rice mills in Burma consume rice meal in the form of gruel.

White rice is graded commercially as Rangoon and Siam, also in accordance with the unbroken condition of the grains as No. 1 quality, or the grain mixed with broken grains as No. 2 quality and so on. Siam rice of the best quality is a long slender grain, almost white and free from dust. Rangoon rice of the best quality is a shorter and more plump grain, white and free from dust.

We should be glad if it could be made clear that in using these trade terms we do so merely as a convenience and with no intention to suggest that the padi produced in Indo-China or Siam is inferior to that produced elsewhere. Misapprehension has arisen in commercial circles as to the significance of these researches and the matter has even been the subject of correspondence between the various Governments. At the local Agricultural Exhibitions *Padi Siam* and *Padi Rangoon* grown locally are exhibited among a host of others. A certain type of grain in commerce has come to be associated with these names and in our use of the terms we do not mean to suggest that they necessarily indicate the country of origin of the rices used in our experiments. With the important questions of the nutritive values of different kinds of padi and the influence thereon of soil, climate, and methods of cultivation we have not concerned ourselves.

Malay or Native rice.

This is prepared from the grain grown by the Malays and the preparation of the rice is almost invariably carried out by the women who prepare it in quantities sufficient for the immediate wants of the household. The grain is sun-dried and transferred in suitable quantities to a wooden mortar fitted with a long wooden pestle. The grain is pounded until the husks are detached and these are removed by winnowing. By repeated pounding and winnowing the husk is entirely removed as well as the pericarp for the most part and the subjacent layers only to a limited extent by attrition and bruising.

The finished product is yellowish and is an admixture of broken and unbroken grains.

By repeated pounding and careful hand-picking it is possible to obtain a fairly white rice free from unbroken grains but the labour entailed is considerable and the natives are usually quite satisfied with a rice from which the entire husk and the most of the pericarp have been removed.

Histology.

In order to compare the different kinds of rice as to their histological characters and in order to examine rices from beri-beri outbreaks for the presence of organisms, it was necessary to obtain entire sections of the grain of sufficient thinness.

The following process was devised and found to yield suitable sections.

The grains are softened for a month or thereabouts in a mucilage composed of:—

| | | | | |
|----------------------------------|-----|-----|-----|--------|
| Gum acacia | ... | ... | ... | 4 pts. |
| Solution of Carbolic Acid (1-20) | ... | ... | ... | 6 „ |

The softened grains are freed from excess of mucilage and imbedded in celloidin. The imbedded grains are placed in alcohol (60%) and after a few days are ready for cutting.

Sections were examined for moulds and for this purpose were stained by the following method :—

1. The sections were stained in anilin-gentian violet solution for from five to ten minutes.
2. The sections were freed from excess of stain and treated with Gram's iodine-solution for one minute.
3. The sections were freed from excess of iodine-solution and washed in absolute alcohol so long as stain continued to come away.
4. The sections were then treated with anilin oil for five minutes. After which the excess of oil was removed and the sections mounted in Canada balsam.

Plates I, II, and III are drawn from actual specimens and reproduce the colouration taken up by the various tissues by this method.

Numerous sections from various kinds of rice were examined in this way. In none of them were moulds or fungi recognised but the method produced excellent differentiation of the tissues and it was easily possible to understand and appreciate the histological differences between padi, parboiled rice, white rice and Malay rice.

A transverse section of a rice grain from which the paleae (husk) have been removed shows three zones (Plate I):—

1. The outer thin pericarp.
2. The layers subjacent to the pericarp, or subpericarpal layers, composed of cells filled with aleurone and fat and comparatively free from starch grains.

3. The remainder of the section, constituting the major part of the section and composed of cells filled with starch grains.

If sections of rice grain be treated with any of the ordinary stains for fat such as Osmic Acid, Sudan III, it will be seen that fat is practically confined to the second zone or subpericarpal layers; a few scattered oil globules may be recognized in the central or starch zone.

Section of a polished rice grain (Plate II) show that the pericarp and most of the subpericarpal layers have been removed. There is usually but a remnant of the fat containing layer and at times not even that remains, the section consisting almost entirely of the starch-containing cells.

Section of parboiled rice (Plate III) which has not been subjected to excessive pearling or polishing show the pericarp mostly removed but the subpericarpal layers practically unaffected.

Sections of Malay rice present an appearance similar to that presented by section of the grain free from husk but the pericarp is removed to a degree the extent of which depends on the amount of pounding and attrition to which the grains have been subjected.

Chemical.

In view of the accumulated mass of evidence which shows that the continuous consumption of rice from which the subpericarpal layers have been removed by polishing causes beri-beri and in view of the histological differences demonstrated between polished and unpolished rices, the suggestion presented itself that by the removal of the subpericarpal layers the grain is deprived of some important nutritive substance or substances.

Analyses were made of the various kinds of rice and for comparison the average results of these analyses are given here:—

| | Protein. | Fat. | Carbohydrate. | Ash. | Moisture. |
|---------------------|----------|------|---------------|------|-----------|
| | % | % | % | % | % |
| Unpolished rice ... | 9.0 | 1.65 | 75.52 | 1.08 | 12.75 |
| Polished rice ... | 7.7 | 0.25 | 77.23 | 0.52 | 14.3 |
| Parboiled rice ... | 7.8 | 0.50 | 76.88 | 0.82 | 14.0 |
| Malay rice ... | 7.3 | 0.63 | 77.19 | 0.88 | 14.0 |

The average daily ration of an oriental labourer contains 1.3 lbs. of rice.

Dealing first with the differences in respect of proteins, that between parboiled and polished rices is small and within the limits of experimental error but admitting the difference to be a real one and assuming all the protein in rice to be available for purposes of nutrition, a person receiving parboiled rice would consume 9 grains more protein than one receiving polished rice and a person receiving Malay rice would consume 36 grains less than the one on polished rice. An explanation on this basis would therefore not be in accordance with the effects produced.

The amount of fat ingested in a ration of 1.3 lbs. of rice would be 45.5 grains in the case of parboiled rice, 22.75 grains in the case of polished rice and 57.25 grains in the case of Malay rice, differences which are appreciable and in accordance with the histological findings but which do not appear to furnish an adequate explanation if the fats be regarded merely as the esters of fatty acids.

The differences in respect of carbohydrates is small, parboiled rice contains more sugar than polished rice or Malay rice but it did not appear that these differences would furnish an explanation.

The ash furnishes a most imperfect idea of the salts contained in rice, all the organic salts being reduced to carbonates but there

is apparently a very constant difference in the amount of ash which is lower in the case of polished rice than in unpolished, parboiled or Malay rices. A person receiving parboiled rice would consume material yielding 74.6 grains of ash and a person receiving polished rice would consume material yielding 47.3 grains of ash. The difference of 27.3 grains is by no means inconsiderable more especially when it is remembered that small amounts of certain inorganic and organic salts have a great importance in the economy.

As we were not in a position to separate out the various salts occurring in rice and it seemed possible then that even if we did accomplish this the results might be unsatisfactory, it was decided to carry out investigations with a view to determining whether a poison or poisons existed in polished rices known to have been associated with outbreaks of beri-beri.

As has been already stated the white and the parboiled rices milled in this country have a common origin in respect of the grain from which they are prepared, therefore any deleterious substance or substances present in the white rice must have developed after polishing and have resulted from the action either of enzymes or of micro-organisms, failure to find these latter notwithstanding.

It has been suggested that the removal of the pericarp and subjacent layers deprives the grain of protective structures and facilitates the action of micro-organisms.

Rice is washed before cooking and the cooking involves exposure to a temperature of 212°F or thereabouts for some time, therefore the ingestion of living organisms with the rice may reasonably be excluded; their activity is confined to the interval which elapses between the milling and the cooking of the rice and the deleterious substance or substances produced by them must be capable of withstanding prolonged exposure to moist heat.

It might be suggested such harmful substances would be removed in the preliminary washing but the extent to which this washing is carried out varies greatly and, not infrequently, it is done in a prefatory manner.

In the initial experiments for the isolation of a poison the white rice was macerated, or boiled and macerated, in water acidulated with a small quantity of Acetic Acid but on account of the rapidity with which moulds developed in the mixture and the difficulty of separating the fluid after maceration these experiments were discontinued.

In the next series of experiments alcohol acidulated with Acetic Acid was employed. The alcoholic extracts were concentrated *in vacuo* and finally freed from alcohol by exposure in evaporating basins to a moderate heat. The extracts were then examined and treated in every way possible for the isolation of poisons but although numerous examinations and experiments were made a poison or poisons were never isolated. The failure to obtain a positive result did not exclude a poison but when it is remembered that a large number of methods were employed it is suggestive that further work along these lines was unlikely to be profitable. We had already noted nutritive differences in the rices which in the light of our then knowledge we did not consider adequate to furnish the explanation sought for but failure to find a satisfactory result on a poison hypothesis caused us to turn our attention to further experiments on the nutritive hypothesis.

Failing chemical methods there remained animal experimentation and it was anticipated that by feeding various animals on rice and rice products, information of value might be obtained.

Feeding Experiments on Animals.

It was designed in the first instance to carry out experiments with anthropoids which we considered were more likely to yield information of value than experiments with animals lower in the zoological scale. Though anthropoids of several species are comparatively common in this country it was not without considerable difficulty that we were enabled to secure a sufficient number of them to make the experiment. Finally six were obtained. These were confined in separate cages and received cooked white rice and water. The results were unsatisfactory, as shortly after the beginning of the experiment several of the animals refused to eat the rice and all of them died of dysentery.

Six monkeys were fed in a similar manner. All of them became very much emaciated and five died after three months apparently of inanition. No degenerative changes were found in the nerves.

These experiments having therefore ended in failure recourse was had to experimentation with fowls, which previous observers had shown to be readily susceptible to a form of polyneuritis following the ingestion of certain foodstuffs.

Eijkman (12) was the first to observe among fowls kept at the Government laboratory at Weltevreden, Java, spontaneous polyneuritis characterised by degeneration of peripheral nerves and atrophy of the ganglion cells in the anterior cornua of the spinal cord. This condition he attributed to feeding with cooked rice. Experimentally he could produce the disease with decorticated, cooked, and raw rice but could prevent it by the addition of the fine inner capsules of the rice grain, the so-called silver layer (*Zilvervliesjes*) and could also cure it in the same way. By the administration of different forms of pure starch a similar disease was produced, potato starch was

the only one that proved to be harmless. Eijkman was of opinion that a toxic substance in rice and the other varieties of starch, developed in the crops of the fowls.

He adds that his investigations show that an apparently physiologically perfect diet can produce severe disease conditions and lead to death.

Eijkman repeated his experiments in Holland and found that the changes in the peripheral nerves were the most important features found on post-mortem examination. They concerned the sensory as well as the motor fibres. They involve bundles of the nerve trunks and present the picture of a non-inflammatory atrophic degeneration such as is seen when a nerve is cut off from its trophic centre. Certain changes in the posterior nerve roots also occur, these likewise show the characters of degeneration and atrophy. The muscles innervated by the affected nerves when treated with osmic acid show a large number of fine fat globules. Feeding with decorticated rice, raw or cooked, and immaterial of the origin or quality of the padi, produced the disease in three or four weeks.

In later investigations Eijkman fed fowls with ground rice and water rolled into balls, and the animals developed polyneuritis. Also in fowls which died after $3\frac{1}{2}$ months fed on husked barley he found many degenerated fibres in the sciatic nerve. If the fowls were fed on unhusked rice that had been heated in a steriliser at 115° or 125°C for two hours the animals developed polyneuritis, after 23 days in the case of rice heated to 115°C and after 21 days in the case of rice heated to 125°C . Simple boiling did not destroy the protective value of unhusked rice. Sterilised barley as well as rye and millet acted in the same way as rice.

Grijns (13) continuing the work of Eijkman found that by adding a certain quantity of Katjang idju (*Phaseolus radiatus*) to a diet of pæled rice the onset of polyneuritis in fowls was prevented.

Hulshoff Pol (14) pursuing the line of research suggested by the experiments of Grijns has shown that Katjang idju has prophylactic and remedial properties in beri-beri. He has further shown that a decoction from a moderately large quantity of Katjang idju possesses the same prophylactic and curative properties as the Katjang idju itself. Dr. Pol considers that an acid ("X" acid) separated from the decoction is the active principle but owing to difficulties in the way of the preparation of this substance no experiments have yet been made with it.

Kiewit de Jonge (15) has also tested the value of Katjang idju as a prophylactic and therapeutic remedy in beri-beri. He carried out an admirably planned and extensive investigation in the Lunatic Asylum at Buitenzorg and fully confirms the work of Grijns and Pol. The very valuable work of the Dutch School will be referred to later in conjunction with the results that have emerged from our own work.

In view of the results already attained by the Dutch physicians it was considered probable fowls would serve our purposes and a preliminary experiment was made by feeding fowls on white rice which was known to have been associated with an outbreak of beri-beri. A control group was fed on parboiled rice.

The fowls were confined in separate cages and were in all respects under identical conditions. The manner of arrangement of the cages is shown in Fig. 1. The cages which were open at the bottom rested on a hard layer of clay the surface of which was covered with sand, and though the possibility of the fowls obtaining such things as worms and the like was not excluded, the conditions were more natural than if the cages had rested on a wooden or concrete floor.

In addition to rice which was supplied twice daily at 10 a.m. and 3 p.m. a small tin filled with water was placed in



I. General View of cages.

each compartment. The original weight of each fowl was noted and thereafter they were weighed once weekly at 12 noon.

A record of each experiment is given in the Tables which follow. The weights of the fowls in each successive week are furnished. The time of development of polyneuritis is indicated and whenever possible the analysis of the actual product supplied in the experiment is noted at the foot of the table.

In the group of fowls on white rice (Experiment No. 1) the first sign of disease was noted in fowl No. 1 on the 26th day of the experiment. In the morning it was observed that there was weakness of the leg muscles and consequent uncertainty of gait. The appearance of the fowl on this day is shown in Fig. II instead of standing upright it reclined on its side in the manner shown. When stimulated it would move about in an uncertain way. Examination of the blood failed to reveal the presence of any parasite or other abnormality.

Two days later the paralysis had advanced very considerably. It was quite unable to walk and the wing muscles were also involved so that these drooped. The diet was now changed to parboiled rice and padi but it was unable to eat and on the 9th day of the illness the fowl died. On post-mortem examination no macroscopic change was noted, there was no effusion into the serous cavities and no marked dilatation of the heart. The principal nerves of the legs and wings were preserved and on subsequent examination shewed characteristic Wallerian degeneration.

To test the possibility of the disease being a communicable one and unconnected with the diet, a fowl (No. 1 of the parboiled rice group) was transferred to the cage occupied by the fowl suffering from polyneuritis on the 5th day of the disease. This fowl was continued on parboiled rice as heretofore and remained healthy at the conclusion of the experiment five weeks later.

Fowl No. 4 developed the disease on the 28th day of the experiment. At 10 a.m. it was noticed to be unsteady but was able to walk about. Two hours later paralysis had extended greatly and the fowl rolled about in the cage in an endeavour to recover the upright position. The head was markedly retracted and when extended immediately returned to this position. The wings were drooping. An attempt was made to feed the fowl with padi but it was unable to eat owing apparently to the spasm of the neck muscles. On the following day the animal died.

This type of case, which was of the convulsive form of polyneuritis of fowls, was not infrequently observed throughout the experiments. It was less common however than the type in which simple paralysis was the principal feature.

On the day of onset of the disease a fowl was transferred from the parboiled rice group and placed in the cage to test the possibility of infection. The result in this as in numerous subsequent tests entirely negatived the possibility of the disease being communicable by contact or through intermediate hosts as ticks or lice.

Within eight weeks, eight of the twelve fowls had developed polyneuritis. Though the experiment was continued for four weeks longer, the other four fowls remained healthy.

The group of fowls on parboiled rice (Experiment No. 2) originally numbered eight. This number was afterwards increased as fowls from this group were transferred to cages occupied by fowls suffering from polyneuritis. In all twelve fowls were under observation for periods varying from five weeks to thirteen weeks. At the conclusion of the experiment all remained healthy. The weights of the fowls in grammes at various stages of the experiment are shown in Table No. 2.



**II. Fowl fed on Siam Rice.
Polyneuritis. First day of disease.**



EXPERIMENT No. 1.—White Rice (No. 2 Siam Depot).

| No. | DESCRIPTION. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. | 6th week. | 7th week. | 8th week. | 9th week. | 10th week. | 11th week. |
|-----|---------------|--------------|-----------|-----------|-----------|--------------|--------------|--------------|-----------|--------------|-----------|------------|------------|
| 1 | Grey hen .. | 1220 | 1247 | 1294 | 1282 | Polyneuritis | | | | | | | |
| 2 | Brown hen .. | 634 | 630 | 607 | 505 | 505 | 498 | 503 | 525 | 500 | 642 | 637 | 621 |
| 3 | Brown hen .. | 848 | 861 | 870 | 807 | 760 | Polyneuritis | | | | | | |
| 4 | Red cock .. | 1280 | 1335 | 1255 | 1132 | 996 | Polyneuritis | | | | | | |
| 5 | Black hen .. | 735 | 690 | 668 | 640 | 572 | 572 | Polyneuritis | | | | | |
| 6 | Red hen .. | 840 | 900 | 878 | 854 | 703 | Polyneuritis | | | | | | |
| 7 | Black cock .. | 637 | 637 | 577 | 560 | 507 | 492 | 455 | 460 | Polyneuritis | | | |
| 8 | White hen .. | 777 | 717 | 555 | 518 | 518 | 555 | 542 | 529 | 535 | 590 | 577 | 660 |
| 9 | Brown hen .. | 1210 | 1177 | 1107 | 1128 | 954 | 957 | 950 | 957 | 942 | 950 | 1015 | 910 |
| 10 | Black cock .. | 864 | 915 | 902 | 910 | 820 | 798 | Polyneuritis | | | | | |
| 11 | Grey cock .. | 822 | 835 | 787 | 715 | 718 | 701 | 688 | 687 | 722 | 740 | 707 | 721 |
| 12 | Red cock .. | 1655 | 1790 | 1735 | 1600 | 1475 | 1466 | Polyneuritis | | | | | |

| Protein. | Fat. | Carbohydrate. | Ash. | Moisture |
|----------|------|-------------------------------|------|----------|
| 7.80 | 0.15 | 77.49 | 0.56 | 14 |
| | | P ₂ O ₅ | | |
| | | 0.28 | | |

EXPERIMENT No. 2.—Parboiled Rice.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. | 8th
week. | 9th
week. | 10th
week. | 11th
week. | 12th
week. | 13th
week. |
|-----|--------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|---------------|---------------|---------------|---------------|
| 1 | Brownish hen | 1295 | 1350 | 1355 | 1345 | 1435 | 1495 | 1465 | 1482 | 1548 | 1548 | | | | |
| 2 | White cock | 1485 | 1565 | 1488 | 1275 | 1514 | 1512 | 1532 | 1568 | 1619 | 1624 | 1635 | 1645 | 1640 | 1662 |
| 3 | Black hen | 625 | 630 | 630 | 640 | 690 | 655 | 672 | 677 | 684 | 672 | | | | |
| 4 | Yellow hen | 1115 | 1155 | 1222 | 1295 | 1417 | 1400 | 1407 | 1375 | 1305 | 1262 | | | | |
| 5 | Grey hen | 700 | 715 | 717 | 702 | 702 | 702 | 724 | 727 | 725 | 752 | | | | |
| 6 | White hen | 625 | 652 | 654 | 646 | 646 | 642 | 657 | 660 | 675 | 693 | | | | |
| 7 | Black cock | 882 | 854 | 848 | 842 | 857 | 807 | 805 | 793 | 748 | 730 | | | | |
| 8 | Brown cock | 865 | 857 | 848 | 804 | 840 | 827 | 842 | 834 | 835 | 855 | | | | |
| 9 | Yellow hen | 1119 | 1120 | 1045 | 1048 | 1037 | 1052 | | | | | | | | |
| 10 | Black hen | 870 | 887 | 877 | 866 | 875 | 877 | 882 | | | | | | | |
| 11 | Brown hen | 1039 | 1007 | 982 | 813 | 840 | 848 | 872 | | | | | | | |
| 12 | Black hen | 837 | 832 | 837 | 872 | 920 | 898 | 908 | 862 | 900 | 882 | 885 | | | |

It was thus shown that fowls were sensitive to differences between these two kinds of rice and the occurrence of polyneuritis furnished a reaction which has proved of the utmost value.

In this as in subsequent experiments every possible effort was made to transmit the disease to the fowls of the control groups but with uniformly negative results.

The previous researches had shown conclusively that white rice was either the vehicle by which the agent producing beri-beri was introduced into the body or that white rice was deficient in some substance or substances essential for metabolism. Parboiled rice neither conveyed this disease producing agent nor was it deficient in the substance or substances essential for metabolism.

By following up the results of these initial experiments on fowls we thought that it might be possible to determine the factors of importance in the etiology of beri-beri. In the case of poisons it should be possible to remove them from white rice and render it innocuous; if it were a deficiency it should be possible to supplement that deficiency either by the addition of the substances removed in the process of polishing or by the addition of substances extracted from parboiled or unpolished rice.

The Poison Hypothesis.

The chemical experiments referred to on page 27 had not been successful in isolating a poison recognizable by chemical means and the inoculation experiments performed on guinea-pigs, rabbits and monkeys with various substances isolated from white rice yielded no satisfactory result.

The disease which occurs among fowls fed on white rice provided a means of carrying out further experiments on the poison hypothesis. The existence of a poison or poisons in white rice had not at this time been definitely excluded and the further possibility remained that the harmful substance or substances could not be detected by any of the chemical reactions employed. Experiments were therefore undertaken with products extracted from white rices. In the first of these (Experiment No. 3) white rice obtained from the supplies issued during the inquiry at Durien Tipus was used. The rice was treated in the following manner :-

1. 1.5 kilogrammes of finely ground rice was macerated for four days in 1.5 litres of 94% alcohol.
2. The mass was then transferred to a percolator and percolated with the alcohol in which it had been macerated.
3. The mass was further percolated with 0.5 litre of 94% alcohol. This operation was repeated three times.
4. After percolation was complete the rice was removed, freed from alcohol by expression, and dried in the sun.

Five fowls were fed on the exhausted rice and three developed polyneuritis within five weeks.

It appeared therefore that percolation with cold alcohol had failed to dissolve out the hypothetical poison.



VI. Fowl fed on Siam Rice which had been extracted with 94 per cent. alcohol.
Polyneuritis. Severe case. Second day of disease.



EXPERIMENT No. 3.—White Rice after extraction with cold 94% alcohol.

| No. | Description | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. | 8th
week. | 9th
week. | 10th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|---------------|
| 1 | Brown cock .. | .. | 887 | 898 | 850 | 795 | 758 | 834 | 805 | 747 | 684 | 592 |
| 2 | Grey hen .. | .. | 947 | 965 | 907 | 893 | 895 | 914 | 938 | 855 | 765 | 724 |
| 3 | Black hen .. | .. | 732 | 712 | 680 | 597 | Polyneuritis | | | | | |
| 4 | Red hen .. | .. | 895 | 845 | 804 | 742 | Polyneuritis | | | | | |
| 5 | Black hen .. | .. | 785 | 750 | 640 | Polyneuritis | | | | | | |

| Protein. | Fat. | Carbohydrate. | Ash. | Moisture. |
|----------|------|-------------------------------|------|-----------|
| 7.3 | 0.14 | 79.68 | 0.48 | 12.4 |
| | | P ₂ O ₅ | | |
| | | 0.27 | | |

EXPERIMENT No. 4.—White Rice after extraction with hot 94% alcohol.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 9th
week. | 10th
week. | 11th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|---------------|---------------|
| 1 | White cock .. | .. | 860 | 782 | 680 | 628 | 595 | 590 | 552 | 520 | Polyneuritis |
| 2 | Red hen .. | .. | 912 | 885 | 815 | 812 | 740 | 730 | 712 | 692 | 678 |
| 3 | Yellow hen .. | .. | 713 | 653 | 555 | 515 | 516 | 492 | 470 | 445 | 427 |
| 4 | Black hen .. | .. | 1275 | 1230 | 1107 | 1037 | 936 | 822 | Polyneuritis | | |

| Protein. | Fat. | Carbohydrate. | Ash. | Moisture. |
|----------|------|-------------------------------|------|-----------|
| 7.0 | 0.11 | 79.01 | 0.48 | 13.4 |
| | | P _a O ₆ | | |
| | | 0.27 | | |

EXPERIMENT No. 5.—Parboiled Rice + Extract from White Rice.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. | 8th
week. | |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--|
| 1 | White cock .. | 1532 | 1568 | 1619 | 1624 | 1635 | 1645 | 1640 | 1662 | 1650 | In 51 days received concentrate
extract from 5400 grammes W. R. |
| 2 | Black hen .. | 872 | 920 | 898 | 908 | 862 | 900 | 860 | 882 | 885 | Do. do. |



In the second experiment (Experiment No. 4) rice from the same source was extracted with hot alcohol in the following manner:—

1. 1.5 kilogrammes of coarsely ground rice had poured over it 1.5 litres boiling 94% alcohol. It was allowed to macerate for several days.

2. The mass was then transferred to an apparatus for extraction with hot alcohol. This was an enlarged form of Soxhlet apparatus made of copper. The rice was treated in this apparatus by extraction with alcohol for 12 hours.

3. 300 c.c. alcohol were added on two occasions, the further extraction being continued for 8 hours.

4. The alcohol was expressed and the rice dried in the sun.

Four fowls were fed on the exhausted rice and two developed polyneuritis. Percolation with hot alcohol had also failed to dissolve out the hypothetical poison.

The bulk of the extracts from the rices had been employed in the chemical experiments but a quantity of the extract prepared by means of hot alcohol remained. This alcoholic extract had most of the alcohol removed from it by distillation *in vacuo* and was finally freed from alcohol by exposure in shallow basins at a low temperature. The alcohol-free extract was emulsified in distilled water and two fowls (Experiment No. 5) fed on parboiled rice received in addition daily an emulsion of the extract representing that obtained from 100 grammes of white rice. The experiment was continued for 51 days. Both fowls gained in weight and showed no signs of any disease.

These experiments showed that no alcohol soluble poison was contained in white rice.

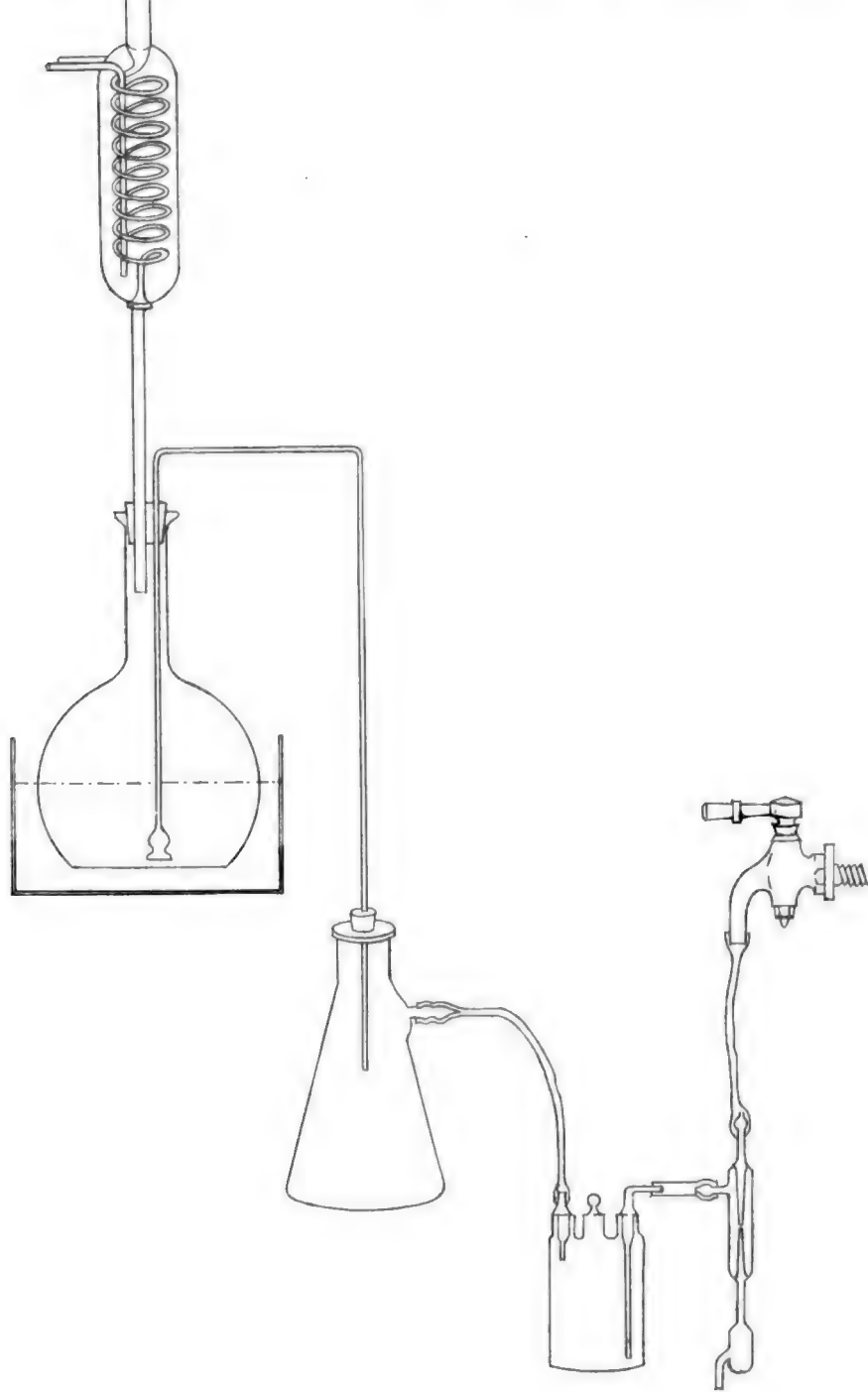
In order to control the results of experiments with products obtained by extracting white rices with alcohol, parboiled rices were treated by precisely similar methods to those already described.

In the first of these experiments (Experiment No. 6) the product employed was obtained by treating parboiled rice with hot alcohol in an extraction apparatus of copper, the copper Soxhlet apparatus already described.

This proved to be an unsatisfactory method for the extraction of parboiled rice and the extraction apparatus shown in the figure was thereafter employed. The bottom of the thistle-head was covered with muslin, the ground rice and alcohol were heated for the requisite period, then by compression of the tube between the flask and the condenser it was attempted to force the fluid over the syphon which once filled would remove the menstruum completely. In practice, this was most troublesome and dangerous but by substituting a filter flask connected with a water-pump the difficulty was overcome and the subsequent extractions of rices were carried out with ease.

The technique employed in the extraction of rice by this method was as follows:—

1. One kilogramme of rice was ground to a coarse powder and placed in a cylindrical jar. One litre of hot alcohol was poured over the rice and the mixture stirred daily for several days.
2. The mixture was transferred to a flask of 2250 cc. capacity and connected up with the extraction apparatus.
3. After heating for one hour the liquid was aspirated off.
4. Half a litre of alcohol was added to the partially exhausted rice, the mixture heated as before and the liquid aspirated off. This process was repeated four times.



EXPERIMENT No. 6.—Parboiled Rice after extraction with hot 94% alcohol.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. | 8th
week. | 9th
week. | 10th
week. |
|-----|---------------|-----------------|--------------|--------------|---------------|--------------|--------------|--------------|--------------|--------------|--------------|---------------|
| 1 | Yellow hen .. | .. | 567 | 560 | 517 | 450 | 425 | 412 | 450 | 448 | 437 | 442 |
| 2 | Black cock .. | .. | 1295 | 1367 | 1260 | 1245 | 1150 | 1150 | 1139 | 1165 | 1125 | 1180 |
| 3 | Black cock .. | .. | 882 | 825 | 745 | 727 | 700 | Polyneuritis | | | | |
| 4 | Black hen .. | .. | 1150 | 1110 | 1012 | 947 | 892 | 925 | 979 | 962 | 965 | 925 |
| | | | | | | | | | | | | |
| | | | Protein. | Fat. | Carbohydrate. | Ash. | Moisture. | | | | | |
| | | | 7.9 | 0.08 | 80.82 | 0.6 | 10.5 | | | | | |

EXPERIMENT No. 7.—Parboiled Rice after extraction with hot 94% alcohol.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th week |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Brown hen .. | .. | 1250 | 1195 | 1035 | 890 | 920 | |
| 2 | Yellow hen .. | .. | 1270 | 1257 | 1255 | 1190 | 1120 | |
| 3 | Black hen .. | .. | 1375 | 1287 | 1160 | 1045 | | Polyneuritis |
| 4 | Yellow cock .. | .. | 1615 | 1550 | 1505 | 1295 | 1305 | |
| 5 | Yellow hen .. | .. | 1257 | 1115 | 1012 | 895 | 800 | Polyneuritis |
| 6 | Black hen .. | .. | 1480 | 1440 | 1350 | 1232 | 1095 | Polyneuritis |
| 7 | White hen .. | .. | 1235 | 930 | 835 | 740 | 655 | |
| 8 | Brown hen .. | .. | 1450 | 1312 | 1200 | 1054 | 905 | Polyneuritis |
| 9 | Yellow hen .. | .. | 1245 | 1140 | 1080 | 1032 | 927 | 850 |
| 10 | Brown hen .. | .. | 1182 | 1154 | 1132 | 1172 | 1172 | 1127 |
| 11 | Yellow hen .. | .. | 1202 | 1075 | 1057 | 1015 | 990 | 985 |
| 12 | Black hen .. | .. | 1188 | 1140 | 1060 | 1005 | 930 | 845 |

EXPERIMENT No. 8.—White Rice and extract from Parboiled Rice.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Brown hen .. | .. | 1365 | 1342 | 1235 | 1235 | 1280 |
| 2 | Red cock .. | .. | 1325 | 1180 | 1135 | 1143 | 1155 |
| 3 | Black cock .. | .. | 1325 | 1180 | 1170 | 1143 | 1145 |
| 4 | Brown cock .. | .. | 1285 | 1262 | 1258 | 1272 | 1260 |
| 5 | Red hen .. | .. | 1185 | 1215 | 1152 | 1112 | 1062 |
| 6 | Black cock .. | .. | 1355 | 1305 | 1372 | 1360 | 1330 |
| 7 | Brown hen .. | .. | 1325 | 1192 | 1205 | 1185 | 1167 |
| 8 | Grey hen .. | .. | 1225 | 1272 | 1295 | 1290 | 1295 |
| 9 | Brown cock .. | .. | 1650 | 1750 | 1760 | 1780 | 1745 |
| 10 | Black hen .. | .. | 1350 | 1348 | 1325 | 1315 | 1290 |
| 11 | Black hen .. | .. | 1343 | 1307 | 1300 | 1305 | 1315 |
| 12 | Brown hen .. | .. | 1240 | 1222 | 1205 | 1227 | 1262 |

The exhausted rice was freed from alcohol by exposure to the sun.

The alcoholic extracts by whichever apparatus obtained, contained a quantity of insoluble matter, due to the coarseness of the method by which separation of the menstruum from the marc was effected. No attempt was made to separate the soluble and insoluble substances from these extracts by further filtration, and it is possible that the presence of insoluble substances in the liquid may explain the equivocal results obtained in the extraction of parboiled rice with 94% alcohol.

In Experiment No. 6 parboiled rice which had been ground and afterwards exhausted several times with hot 94% alcohol was used. It was anticipated that by this method such substances as lecithin and a portion of other fats would be removed from it. Four fowls only were employed for the experiment. In the fifth week one of them, No. 3, developed a condition indistinguishable clinically from other cases of polyneuritis in fowls fed on white polished rice.

This result indicated that some protective substance ordinarily present in parboiled rice had been removed by treatment with hot 94% alcohol and further experiments were undertaken to test the value of this suggestion.

In the experiments next to be described alcohol of 94% strength was employed. For the products employed in Experiments Nos. 7 and 8 an apparatus similar to that illustrated by the figure was employed. Here, however, a positive pressure generated by the boiling fluids within the flask was employed for withdrawing the liquid. The compact character of the rice mass made it difficult to secure more than very coarse filtration of the solution. Of twelve fowls fed on the exhausted rice in Experiment No. 7 two developed polyneuritis within five weeks and two more in the sixth week. Fowls fed on white rice to

which was added the extract from 83 grammes of unpolished parboiled rice daily (Experiment No. 8) all remained healthy.

It was concluded from these experiments that the protective substances were soluble in hot alcohol of 94% strength.

A further experiment, No. 9, was carried out with unpolished parboiled rice extracted with 94 % alcohol in the apparatus illustrated. One fowl died in the seventh week. No cases of polyneuritis occurred.

In the control experiment, No. 10, in which the extracted substances were added to a white rice diet, all the fowls remained healthy.

These latter results can only be explained by assuming that the essential substances were only partially extracted from the unpolished parboiled rice.

The next experiment, No. 11, was one in which parboiled rice from the same sample as used in Experiment No. 6 was treated with cold alcohol of 94% strength by maceration and percolation. At first five fowls only were employed in this group but as cases of polyneuritis occurred in fowls fed on white rice treated by identical methods, fowls were transferred to the latter group and continued on the same food to test the possibility of conveying infection. To replace these, fowls were added and in the course of the experiment eight fowls in all were under observation.

From an examination of the chart it will appear that while some of the fowls lost weight the general result was a gain in weight and all remained healthy at the conclusion of the experiment.

It would appear that cold 94% alcohol is not an effective solvent of the protective substances.

EXPERIMENT No. 9.—Parboiled Rice after extraction with 94% alcohol.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|----------------------------------|
| 1 | Black hen .. | 815 | 772 | 795 | 780 | 795 | 762 | 815 | 810 |
| 2 | Brown hen .. | 895 | 832 | 820 | 820 | 775 | 745 | 755 | 745 |
| 3 | Yellow hen .. | 1070 | 1100 | 1035 | 955 | 975 | 960 | 1015 | 1005 |
| 4 | Brown hen .. | 975 | 952 | 940 | 865 | 845 | 825 | 850 | 820 |
| 5 | Yellow hen .. | 1100 | 1115 | 1050 | 1005 | 1020 | 937 | 950 | 935 |
| 6 | Yellow hen .. | 960 | 960 | 920 | 875 | 860 | 845 | 855 | 850 |
| 7 | Red cock .. | 1075 | 1010 | 1045 | 1000 | 975 | 880 | 815 | 735 |
| 8 | White hen .. | 820 | 810 | 810 | 800 | 800 | 775 | 820 | 810 |
| 9 | White hen .. | 890 | 912 | 920 | 870 | 840 | 790 | 770 | 720 |
| 10 | Yellow hen .. | 1075 | 1080 | 1045 | 1050 | 1055 | 982 | 985 | 955 |
| 11 | Brown hen .. | 990 | 1000 | 1010 | 1015 | 970 | 930 | 925 | 905 |
| 12 | Brown hen .. | 1290 | 1110 | 1125 | 1060 | 955 | 915 | 815 | Died. No degeneration of nerves. |

| Protein. | Fat. | Carbohydrate. | Ash. | Moisture. |
|----------|------|-------------------------------|------|-----------|
| 7.60 | 0.06 | 80.66 | 0.88 | 10.80 |
| | | P ₂ O ₅ | | |
| | | 0.425 | | |

EXPERIMENT No. 10.—White Rice + Extract from Parboiled Rice.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. |
|------------|---------------|-----------------|--------------|-------------------------------|--------------------------------|--------------|--------------|--------------|--------------|
| 1 | Yellow hen .. | 1092 | 1150 | 1120 | 1020 | 1010 | 1000 | 1000 | 970 |
| 2 | Brown hen .. | 1065 | 1052 | 1010 | 935 | 905 | 900 | 905 | 910 |
| 3 | White hen .. | 1272 | 1185 | 1200 | 1180 | 1170 | 1215 | 1200 | 1210 |
| 4 | Brown hen .. | 1048 | 1085 | 1085 | 1005 | 920 | 955 | 975 | 960 |
| 5 | White hen .. | 1315 | 1295 | 1230 | 1220 | 1232 | 1220 | 1255 | 1245 |
| 6 | Brown hen .. | 1340 | 1260 | 1275 | 1280 | 1277 | 1265 | 1290 | 1220 |
| 7 | Brown cock .. | 1415 | 1390 | 1425 | 1350 | 1590 | 1590 | 1570 | 1385 |
| 8 | Red cock .. | 1495 | 1085 | Died | Cause of death not determined. | | | | |
| 9 | White hen .. | 1450 | 1480 | 1425 | 1285 | 1245 | 1255 | 1265 | 1220 |
| 10 | Grey hen .. | 1260 | 1130 | 1175 | 1215 | 1280 | 1275 | 1280 | 1215 |
| | | | | | | | | | |
| White Rice | | Protein. | Fat. | Carbohydrate. | Ash. | Moisture. | | | |
| | | 6.90 | .20 | 77.26 | 0.40 | 15.24 | | | |
| | | | | P ₂ O ₅ | | | | | |
| | | | | 0.22 | | | | | |

EXPERIMENT No. 11.—Parboiled Rice after extraction with cold 94% alcohol.

| No. | DESCRIPTION. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. | 6th week. | 7th week. | 8th week. | 9th week. | 10th week. |
|-----|----------------|--------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|--------------|------------|
| 1 | Black hen .. | 852 | 827 | 815 | 770 | 735 | 715 | 687 | 648 | 552 | 475 | 445 |
| 2 | Red hen .. | 875 | 910 | 930 | 1007 | 1019 | 1037 | 1012 | 1002 | 988 | 965 | 940 |
| 3 | Red hen .. | 804 | 820 | 837 | 822 | 837 | 835 | 867 | 852 | 870 | Transferred. | |
| 4 | Yellow cock .. | 1097 | 1137 | 1208 | 1210 | 1287 | 1298 | 1315 | 1325 | 1345 | 1402 | |
| 5 | Black hen .. | 625 | 640 | 609 | 595 | 577 | 595 | 572 | 598 | 605 | 655 | |
| 6 | Yellow hen .. | 900 | 889 | 844 | 846 | 798 | 763 | 705 | | | | |
| 7 | Red cock .. | 1208 | 1184 | 1230 | 1188 | 1165 | 1074 | 1032 | | | | |
| 8 | Red hen .. | 874 | 875 | 874 | 895 | 890 | 882 | 850 | | | | |

| Protein. | Fat. | Carbohydrate. | Ash. | Moisture. |
|----------|------|---------------|------|-----------|
| 8.0 | 0.06 | 78.0 | 0.78 | 12.7 |

EXPERIMENT No. 12.—Parboiled Rice after extraction with hot Proof Spirit.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Black cock .. | 1490 | 1350 | 1250 | 1150 | 1030 | 935 | |
| 2 | Black hen .. | 1490 | 1475 | 1340 | 1300 | 1240 | 1190 | |
| 3 | Brown cock .. | 1275 | 1235 | 1140 | 1110 | 1030 | Polyneuritis | |
| 4 | Yellow hen .. | 1210 | 1245 | 1165 | 1095 | 1005 | 925 | |
| 5 | Yellow hen .. | 1130 | 980 | 935 | 835 | 740 | 665 | |
| 6 | Red cock .. | 1390 | 1355 | 1250 | 1080 | Polyneuritis | | |
| 7 | White hen .. | 1435 | 1370 | 1280 | 1190 | 1160 | 1060 | |
| 8 | White cock .. | 1465 | 1325 | 1180 | 1080 | 995 | 910 | |
| 9 | Yellow hen .. | 1335 | 1217 | 1080 | 985 | 855 | 890 | |
| 10 | Black cock .. | 1560 | 1420 | 1325 | 1155 | Polyneuritis | | |
| 11 | Yellow hen .. | 1390 | 1325 | 1255 | 1190 | 1130 | 1095 | |
| 12 | White cock .. | 1475 | 1285 | 1170 | 1080 | 985 | Polyneuritis | |

| | Protein. | Fat. | Carbohydrate. | Ash. | Moisture. |
|--|----------|------|-------------------------------|------|-----------|
| | 7.65 | 0.10 | 80.01 | 0.44 | 11.8 |
| | | | P ₂ O ₅ | | |
| | | | 0.29 | | |

EXPERIMENT No. 13.
White Rice + Proof Spirit extract from Parboiled Rice.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | 1195 | 1160 | 1180 | 1215 | 1175 | 1182 |
| 2 | Red cock .. | 1320 | 1312 | 1310 | 1310 | 1310 | 1292 |
| 3 | Red cock .. | 1122 | 1060 | 1050 | 1052 | 1050 | 1030 |
| 4 | Yellow cock .. | 1315 | 1320 | 1310 | 1385 | 1410 | 1390 |
| 5 | Red cock .. | 1420 | 1390 | 1425 | 1395 | 1370 | 1342 |
| 6 | Yellow cock .. | 1700 | 1710 | 1715 | 1740 | 1725 | 1722 |
| 7 | White hen .. | 1820 | 1690 | 1660 | 1710 | 1780 | 1740 |
| 8 | Grey hen .. | 1870 | 1910 | 1935 | 1760 | 1840 | 1930 |
| 9 | Red cock .. | 1710 | 1705 | 1687 | 1740 | 1765 | 1770 |
| 10 | Yellow cock .. | 1640 | 1650 | 1650 | 1660 | 1660 | 1655 |

In Experiments No. 12 and 13 unpolished parboiled rice was extracted with alcohol of proof spirit strength in the apparatus figured. The extracts freed from alcohol in the manner already described were used in these experiments.

In Experiment No. 12 twelve fowls were fed on the exhausted rice; three developed polyneuritis within five weeks. In the corresponding experiment, No. 13, in which the alcohol-free extract obtained from 60 grammes of unpolished parboiled rice was added daily to the diet of twelve fowls on white polished rice, all the fowls remained healthy for five weeks.

This result shows that the essential protective substances are also soluble in alcohol of proof spirit strength.

In the foregoing series of experiments, which were of a tentative nature, no evidence was obtained that white (polished) rice ever contained a beri-beri producing agent. The results taken in conjunction with the histological findings suggested that by the removal of the subpericarpal layers of the grain in the process of milling, the grain is deprived of some substance or substances of high physiological value.

In subsequent experiments this conclusion was abundantly confirmed. In giving a detailed record of these experiments therefore, these are arranged in accordance with the conclusions ultimately arrived at and not in chronological order. It is hoped that in this way the position will be made clearer.

Experiments with various White Rices.

Experiments were carried out with white polished rices from various sources. These may properly be grouped in a single series.

In the first experiment of the series (Experiment No. 14) the white polished rice used in the inquiry at Durian Tipus was employed. Of the four fowls used for the experiment three developed polyneuritis within four weeks.

The second experiment of the series (Experiment No. 15) was one designed to test the effect of feeding fowls on a white polished rice while at the same time another group (Experiment No. 25) was fed on the original partially husked padi from which this white rice had been prepared, and yet another (Experiment No. 32) on the white polished rice to which the substances removed in the polishing process had been added. In the group under review, those to which white rice alone was given, one fowl (No. 8) died on the third week without signs of polyneuritis—six others developed polyneuritis within five weeks.

The third experiment of the series (Experiment No. 16) was carried out to test whether, as alleged by Braddon, rice that had become stale on account of changes occurring in it in the interval between milling and consumption was more harmful in its influence than freshly milled rice. An assistant was stationed in Singapore who forwarded to the laboratory daily by the most expeditious route a quantity of white polished rice milled on the day of despatch. Of twelve fowls fed on this rice five developed typical polyneuritis within four weeks, a result similar to that in experiments in which white polished rice varying in age from a few months to two years was employed.



III. Fowl fed on Siam Rice
Polyneuritis. Mild case. Eighth day of disease.



IV. Fowl fed on Siam Rice.
Polyneuritis. Mild case. Fifth day of disease.

EXPERIMENT No. 14.—White Rice (from Beri-beri outbreak Durien Tipus).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. |
|----------|----------------|-----------------|-------------------------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Black cock .. | 2098 | 2144 | 1892 | 1915 | Polyneuritis | | |
| 2 | White hen .. | 1292 | 1222 | 1090 | 960 | 817 | 667 | 672 |
| 3 | Yellow cock .. | 1439 | 1300 | 1080 | 969 | Polyneuritis | | |
| 4 | Brown hen .. | 1090 | 1020 | 952 | 880 | Polyneuritis | | |
| Protein. | | Fat. | Carbohydrate. | | Moisture | Ash. | | |
| 7.45 | | 0.17 | 78.02 | | 13.85 | 0.51 | | |
| | | | P ₂ O ₅ | | | | | |
| | | | 0.28 | | | | | |

EXPERIMENT 15.—White Rice (No. 2 Siam milled in Singapore).

| No. | DESCRIPTION. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. |
|-----|---------------|--------------|-----------|-----------|-----------------------|--------------|--------------|
| 1 | Yellow hen .. | 1052 | 1035 | 1050 | 1072 | 1090 | 1060 |
| 2 | White hen .. | 1215 | 1265 | 1187 | Polyneuritis | | |
| 3 | Red cock .. | 1555 | 1452 | 1295 | 1115 | Polyneuritis | |
| 4 | Black hen .. | 1300 | 1240 | 1115 | 1012 | 880 | Polyneuritis |
| 5 | Brown hen .. | 882 | 807 | 800 | 770 | 785 | 755 |
| 6 | Black hen .. | 872 | 830 | 905 | 905 | 862 | 820 |
| 7 | Black cock .. | 855 | 802 | 785 | 692 | Polyneuritis | |
| 8 | Brown cock .. | 730 | 650 | 545 | Died. No Polyneuritis | | |
| 9 | Yellow hen .. | 1200 | 1110 | 985 | 865 | 768 | Polyneuritis |
| 10 | White hen .. | 1440 | 1465 | 1248 | 1075 | 982 | Polyneuritis |
| 11 | White hen .. | 1470 | 1415 | 1380 | 1192 | 1095 | 1030 |
| 12 | White hen .. | 1178 | 1185 | 1032 | 945 | 860 | 875 |

| Protein. | Fat. | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 7.3 | 0.40 | 77.43 | 14.3 | 0.57 |
| | | P ₂ O ₅ | | |
| | | 0.28 | | |

EXPERIMENT No. 16.—White Rice (recently milled).

| No. | DESCRIPTION. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. |
|----------|-----------------|--------------|-------------------|-----------|-----------|-----------|--------------|
| 1 | Red cock .. | 1570 | 1500 | 1320 | 1255 | | Polyneuritis |
| 2 | White hen .. | 810 | 830 | 795 | 757 | 725 | |
| 3 | Brownish hen .. | 1327 | 1138 | 1048 | 935 | 725 | |
| 4 | Black hen .. | 815 | 815 | 732 | 717 | | Polyneuritis |
| 5 | Brown hen .. | 1250 | 1207 | 1088 | 955 | | Polyneuritis |
| 6 | Red cock .. | 1135 | 1102 | 982 | | | Polyneuritis |
| 7 | White hen .. | 895 | 875 | 777 | 657 | 685 | |
| 8 | Yellow cock .. | 1100 | 1020 | 960 | 892 | 855 | |
| 9 | Grey hen .. | 1173 | 1175 | 1080 | 935 | 815 | |
| 10 | Black cock .. | 1225 | 1203 | 1098 | 930 | | Polyneuritis |
| 11 | White cock .. | 1010 | 890 | 762 | 632 | 545 | |
| 12 | Yellow hen .. | 892 | 822 | 750 | 684 | 591 | |
| Protein. | | Fat. | Carbohydrate. | Ash. | Moisture. | | |
| 8.1 | | 0.41 | 76.76 | 0.47 | 14.26 | | |
| | | | P, O ₂ | | | | |
| | | | 0.27 | | | | |

EXPERIMENT No. 17.—White Rice (No. 1 Siam).

| No. | Description. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. | 6th week. | 7th week. | 8th week. | 9th week. |
|-----|----------------|--------------|-----------|-----------|-----------|--------------|--------------|-----------|--------------|-----------|--------------|
| 1 | Red cock .. | 1385 | 1370 | 1342 | 1382 | 1365 | Polyneuritis | | | | |
| 2 | Brown hen .. | 1045 | 1075 | 1060 | 1107 | 1050 | 965 | 895 | 945 | 980 | 1015 |
| 3 | Yellow cock .. | 1040 | 1027 | 987 | 1060 | 1032 | Polyneuritis | | | | |
| 4 | Grey hen .. | 1095 | 1115 | 1045 | 1032 | 965 | 930 | 995 | 940 | 965 | Polyneuritis |
| 5 | Red hen .. | 1070 | 1052 | 1045 | 975 | 930 | 907 | 935 | 950 | 975 | 990 |
| 6 | Yellow hen .. | 1435 | 1418 | 1305 | 1222 | 1177 | Polyneuritis | | | | |
| 7 | Yellow hen .. | 1230 | 1180 | 1102 | 1037 | 990 | 940 | 940 | 900 | 880 | 920 |
| 8 | Brown hen .. | 1645 | 1625 | 1505 | 1465 | 1390 | 1257 | 1215 | Polyneuritis | | |
| 9 | Yellow cock .. | 2035 | 2080 | 1900 | 1845 | Polyneuritis | | | | | |
| 10 | Red cock .. | 1550 | 1635 | 1497 | 1480 | 1435 | Polyneuritis | | | | |
| 11 | White cock .. | 1425 | 1440 | 1337 | 1335 | Polyneuritis | | | | | |
| 12 | Red cock .. | 1550 | 1608 | 1495 | 1440 | Polyneuritis | | | | | |

| Protein. | Fat. | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 6.9 | 0.20 | 77.26 | 15.24 | 0.40 |
| | | P ₂ O ₅ | | |
| | | 0.22 | | |

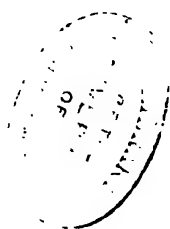
EXPERIMENT No. 18.—White Rice (No. 1 Siam).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Brown hen .. | 1290 | 1185 | 1175 | 1160 | 1182 | Polyneuritis | Polyneuritis |
| 2 | Red cock .. | 1190 | 1160 | 1150 | 1125 | Polyneuritis | Polyneuritis | |
| 3 | Black cock .. | 1140 | 1180 | 1145 | 1145 | Polyneuritis | Polyneuritis | |
| 4 | Brown cock .. | 1275 | 1210 | 1145 | Polyneuritis | Polyneuritis | | |
| 5 | Red hen .. | 1045 | 940 | 892 | 770 | Polyneuritis | Polyneuritis | |
| 6 | Black cock .. | 1310 | 1270 | 1220 | 1222 | Polyneuritis | Polyneuritis | |
| 7 | Brown hen .. | 1185 | 1195 | 1180 | 1060 | 955 | Polyneuritis | Polyneuritis |
| 8 | Grey hen .. | 1255 | 1155 | 1055 | 990 | 990 | 1035 | 1050 |
| 9 | Brown cock .. | 1820 | 1780 | 1720 | 1690 | Polyneuritis | Polyneuritis | |
| 10 | Black hen .. | 1210 | 1170 | 1132 | 992 | Polyneuritis | Polyneuritis | |
| 11 | Black hen .. | 1300 | 1290 | 1262 | 1152 | Polyneuritis | Polyneuritis | |
| 12 | Brown hen .. | 1410 | 1415 | 1355 | 1332 | 1282 | Polyneuritis | Polyneuritis |

EXPERIMENT No. 19.

White Rice (washed thoroughly before feeding).

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Brown cock .. | 1575 | 1455 | 1350 | 1330 | Polyneuritis |
| 2 | White hen .. | 1430 | 1350 | 1180 | 1060 | |
| 3 | Black hen .. | 1385 | 1315 | 1190 | 1150 | |
| 4 | Red cock .. | 1505 | 1450 | 1280 | Polyneuritis | |
| 5 | White hen .. | 1575 | 1430 | 1310 | 1220 | |
| 6 | Brown hen .. | 1325 | 1270 | 1090 | 1015 | |
| 7 | Black hen .. | 1390 | 1310 | 1130 | 1020 | |
| 8 | Red cock .. | 1930 | 1790 | 1485 | Polyneuritis | |
| 9 | Brown hen .. | 1100 | 1055 | 965 | 895 | |
| 10 | Black hen .. | 1100 | 1035 | 980 | 975 | |
| 11 | White hen .. | 1530 | 1415 | 1360 | 1205 | |
| 12 | Brown cock .. | 1415 | 1340 | 1235 | 1180 | |



EXPERIMENT No. 20.—White Rice (No. 1 Siam).

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Brown cock .. | 1135 | 1155 | 1160 | 1055 | Polyneuritis |
| 2 | Yellow cock .. | 1280 | 1230 | 1165 | 1010 | Polyneuritis |
| 3 | Black cock .. | 1180 | 1200 | 1225 | 1155 | Polyneuritis |
| 4 | Brown cock .. | 1150 | 1145 | 1170 | 1105 | 1050 |
| 5 | Black cock .. | 1175 | 1200 | 1100 | 985 | Polyneuritis |
| 6 | Yellow cock .. | 1090 | 990 | 955 | 910 | 780 |





Wallerian degeneration.
Teased preparation from Sciatic nerve of fowl suffering from Polyneuritis.

Experiments Nos. 17 and 18 were carried out with white polished rice of the best quality (No. 1 Siam) purchased locally. In one of them nine fowls out of twelve developed polyneuritis within nine weeks, seven cases occurring before the end of the fifth week. In the other, eleven cases among twelve fowls occurred within five weeks. It appeared in these experiments that certain fowls were more resistant than others to the injurious influence of white rice. The reason for this was not ascertained though the observation was made that such fowls were commonly the younger members of the group, while those who succumbed most quickly were the full grown fowls who consumed a large quantity of rice. It was also noted that after two or three weeks fowls on white rice usually showed a disinclination to eat.

These observations which are similar to those made when human beings were under observation appear to us to be irreconcilable with the idea of simple deprivation of proteins, fats or carbohydrates being the final explanation of the occurrence of the disease.

Experiment No. 19 was carried out with a white polished rice as a control to other experiments in which this same sample was employed as the principal article of diet, various materials being added to it. Experiment No. 20 was carried out for a similar purpose.

In every case of polyneuritis Wallerian degeneration (Photo) was demonstrated microscopically in the affected nerves. The changes are apparently identical in every way with those found in the peripheral nerves in beri-beri. An extensive investigation of all the materials obtained is now being carried out by Dr. Fletcher.

Rangoon Rice.

There is evidence that the variety of white polished rice known as Rangoon rice is less harmful than Siam rice. Braddon

quotes examples in support of this view and instances have fallen under our own observation.

In Experiment No. 21 Rangoon rice was employed. Fowls No. 10 and No. 12 died in the 5th and 4th week respectively but there was no clinical or pathological evidence that they suffered from polyneuritis. Both had a form of purulent conjunctivitis.

No. 4 (Fig. 5) was a typical case of polyneuritis in the 5th week and no other case occurred until the 15th week when Nos. 7 and 8 suffered from it. All three cases resulted fatally and the diagnosis was confirmed post-mortem.

Such evidence as is furnished by this single experiment confirms the experience of those who assert the comparative harmlessness of Rangoon rice.

The explanation of this observation is a matter for further inquiry. Whether the padi grown in Burmah is richer in protective substances and therefore can afford to lose in the polishing process a greater quantity of them without harmful result or whether there is some important difference in the milling process it is impossible yet to say. From histological examination of the grains we incline to the former view.



**V. Fowl fed on Rangoon Rice.
Polyneuritis. Severe case. First day of disease.**

EXPERIMENT No. 21.—White Rice (Rangoon).

| No. | Description. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. | 6th week. | 7th week. | 8th week. | 9th week. | 10th week. | 11th week. | 12th week. | 13th week. | 14th week. | 15th week. |
|-----|---------------|--------------|-----------|-----------|-----------|-----------|--------------|-----------|-----------|-----------|-----------|------------|-----------------|------------------|------------|------------|--------------|
| 1 | Red cock .. | 1104 | 1147 | 1132 | 1175 | 1174 | 1197 | 1192 | 1237 | 1185 | 1210 | 1190 | 1195 | 1202 | 1248 | 1185 | 1205 |
| 2 | White hen .. | 857 | 888 | 854 | 810 | 830 | 822 | 818 | 765 | 799 | 788 | 762 | 760 | 715 | 658 | 545 | Died |
| 3 | Black hen .. | 945 | 940 | 905 | 857 | 788 | 757 | 735 | 700 | 682 | 630 | 554 | 480 | Died | | | |
| 4 | Yellow hen .. | 1227 | 1207 | 1150 | 995 | 850 | Polyneuritis | | | | | | | | | | |
| 5 | Black cock .. | 1280 | 1285 | 1327 | 1262 | 1293 | 1307 | 1352 | 1248 | 1282 | 1280 | 1250 | 1240 | 1252 | 1255 | 1215 | 1155 |
| 6 | Black hen .. | 1225 | 1252 | 1252 | 1265 | 1322 | 1425 | 1492 | 1471 | 1529 | 1395 | 1398 | 1440 | 1422 | 1352 | 1245 | 1170 |
| 7 | Red cock .. | 957 | 955 | 910 | 885 | 952 | 922 | 898 | 829 | 787 | 730 | 705 | 660 | 655 | 632 | 607 | Polyneuritis |
| 8 | White cock .. | 745 | 769 | 765 | 662 | 625 | 639 | 622 | 602 | 565 | 539 | 535 | 530 | 512 | 485 | 450 | Polyneuritis |
| 9 | Grey hen .. | 667 | 672 | 667 | 675 | 693 | 707 | 700 | 675 | 712 | 677 | 691 | 737 | 734 | 747 | 740 | 735 |
| 10 | Black cock .. | 729 | 757 | 657 | 548 | 465 | Died | | | | | | | | | | |
| 11 | Grey hen .. | 637 | 637 | 632 | 595 | 614 | 552 | 489 | 439 | 467 | 479 | 460 | 445 | 430 | 422 | 409 | 395 |
| 12 | Black cock .. | 845 | 852 | 747 | 595 | Died | | | | | | | | | | | |
| 13 | Red cock .. | 1239 | 1127 | 1140 | 1180 | 1177 | 1185 | 1205 | 1210 | 1220 | 1185 | 1120 | Replaced No. 4. | | | | |
| 14 | Brown hen .. | 1032 | 1045 | 1077 | 1087 | 1154 | 1160 | 1215 | 1230 | 1245 | 1252 | 1190 | 1100 | Replaced No. 10. | | | |
| 15 | Brown hen .. | 922 | 952 | 983 | 970 | 975 | 1004 | 998 | 1000 | 995 | 965 | 942 | 860 | Replaced No. 12. | | | |

| | | | | | | | | | |
|----------|-----|------|------|-------------------------------|-------|-----------|-------|------|------|
| Protein. | 7.3 | Fat. | 0.63 | Carbohydrate. | 77.15 | Moisture. | 14.18 | Ash. | 0.74 |
| | | | | P ₂ O ₅ | 0.33 | | | | |

EXPERIMENT No. 22.—Simple Starvation.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Brown hen .. | 1170 | 1052 | 960 | 868 | 842 | |
| 2 | Yellow hen .. | 1365 | 1230 | 1115 | 1070 | 1024 | |
| 3 | Black hen .. | 1085 | 940 | 859 | 782 | 750 | |
| 4 | Yellow cock .. | 1490 | 1335 | 1225 | 1122 | 1070 | |

EXPERIMENT No. 23.—Simple Starvation.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | — |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|---|
| 1 | Yellow hen .. | 1612 | 1500 | 1355 | 1275 | 1200 | | Killed for examination of
nerves 5th week. No
degeneration. |
| 2 | Yellow cock .. | 1285 | 1170 | 1030 | 930 | 815 | | |
| 3 | Yellow hen .. | 1860 | 1790 | 1615 | 1490 | 1400 | | |
| 4 | Red cock .. | 1470 | 1385 | 1190 | 925 | | | Killed for examination of
nerves 4th week. No
degeneration. |
| 5 | Brown hen .. | 1010 | 915 | 835 | 760 | 675 | | |
| 6 | Red cock .. | 1420 | 1325 | 1120 | 930 | 720 | | Died 5th week. No
degeneration of nerves. |
| 7 | Yellow hen .. | 1350 | 1270 | 1220 | 1100 | 1000 | | |
| 8 | Red cock .. | 1720 | 1640 | 1455 | 1350 | 1245 | | |
| 9 | Black hen .. | 1850 | 1765 | 1615 | 1500 | 1395 | | Died 4th week. No de-
generation of nerves. |
| 10 | Red cock .. | 1360 | 1280 | 1150 | 1035 | 950 | | |
| 11 | Red cock .. | 1430 | 1345 | 1090 | 880 | | | |
| 12 | Yellow hen .. | 1510 | 1400 | 1237 | 1155 | 1100 | | |

Starvation and Forced Feeding Experiments.

As has been already stated it was usually found that after the first few weeks on a white rice diet, most fowls showed a disinclination to eat. Commonly also fowls lost greatly in weight before the development of polyneuritis. That these were by no means constant features however will be seen on reference to the tables which show that in some cases fowls continued to eat well and maintain their normal weight until the disease declared itself.

Despite the fact that groups of fowls on a diet of padi unpolished rice or parboiled rice usually continued to eat well throughout, the suggestion was raised that on account of the monotonous nature of a diet of white rice the fowls ceased to eat and that polyneuritis was a result of partial simple starvation. This was at all events a point that required to be tested and accordingly experiments were carried out.

In the first experiment of this series (Experiment No. 22) four fowls hitherto feeding on padi were employed. Nothing but water was supplied. The bottom of the cage being open they had access to such food, insects and the like, as could be obtained there—this condition of affairs was, however, controlled by the fact that all fowls including those on white rice were similarly circumstanced. Within four weeks all the fowls had lost greatly in weight and were weak but none of the signs constantly associated with polyneuritis had shown themselves. Within a similar period some fowls of a group fed on white polished rice had invariably shown signs of polyneuritis.

The number of fowls in the first experiment was of course too small to furnish any definite information and a second experiment with 12 fowls was begun (Experiment No. 23).

Within five weeks two of the fowls composing this group had died and two others were killed when their condition was such that it was apparent they would die within a few hours. None had showed any clinical signs of polyneuritis and careful examination of the nerves showed no characteristic degeneration changes.

A third experiment, No. 24, was carried out with a similar result.

It was concluded that polyneuritis of fowls is not due to general deprivation of food but to deprivation of some particular element in a diet of white polished rice.

In order to test further the correctness of this conclusion Dr. Fletcher carried out an experiment in which two groups of fowls were fed on white rice (Experiment No. 25).

To the first group (Fowls Nos. 1—6) in the following table 30 grammes of white rice was supplied twice daily in the ordinary way. To the second group Nos. 7—12, 30 grammes of white rice was passed into the crop twice daily.

The result showed that there was no difference between the two groups as to their liability to develop polyneuritis. Polyneuritis therefore cannot be due to simple deprivation of food.

An interesting observation made by Dr. Fletcher in connexion with this experiment was that when a fowl which was being forcibly fed suffered from the disease the normal digestive powers were markedly interfered with and the crop remained constantly distended with rice. If now a small quantity of polishings was passed into the crop the accumulated rice seemed to be dissolved and normal digestive power was speedily regained.

EXPERIMENT No. 24.—Simple Starvation.

| No. | Description. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | --- |
|-----|---------------|--------------|-----------|-----------|-----------|-----------|--|
| 1 | Black hen .. | 915 | 755 | 645 | 525 | Died. | |
| 2 | Brown hen .. | 1630 | 1370 | 1170 | 955 | Died. | |
| 3 | Black hen .. | 1225 | 1070 | 980 | 845 | 708 | |
| 4 | Brown hen .. | 1125 | 970 | 815 | 715 | 545 | |
| 5 | White cock .. | 1775 | 1530 | 1380 | 1295 | 1130 | (Killed in 5th week.
Normal except globulation
of few nerve fibres. |
| 6 | Brown hen .. | 1000 | 830 | 740 | 660 | 575 | |
| 7 | Brown hen .. | 1505 | 1325 | 1210 | 1115 | 1020 | |
| 8 | Brown cock .. | 1440 | 1210 | 1060 | 925 | 655 | |
| 9 | Yellow hen .. | 1155 | 970 | 870 | 765 | 550 | |
| 10 | Yellow hen .. | 1160 | 990 | 895 | 825 | 755 | |

EXPERIMENT No. 25.—White Rice. Nos. 1—6 Ordinary Feeding.
Nos. 7—12 Forced Feeding.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|--------------------------|-----------------|--------------|--------------|--------------|-------------------|
| 1 | Red and black cock .. | 1405 | 1320 | 1200 | | Polyneuritis |
| 2 | White and black cock .. | 1255 | 1190 | 1070 | | Polyneuritis |
| 3 | Spotted cock .. | 1620 | 1772 | 1680 | 1540 | |
| 4 | Yellow and white cock .. | 1385 | 1385 | 1250 | 1180 | |
| 5 | Red and black cock .. | 1285 | 1260 | 1050 | 730 | Polyneuritis |
| 6 | Red and black cock .. | 1340 | 1205 | 935 | | Polyneuritis |
| 7 | White and brown cock .. | 1000 | 1000 | 950 | | Polyneuritis |
| 8 | Red and black cock .. | 1635 | 1630 | 1570 | | Polyneuritis |
| 9 | Black and red cock .. | 1485 | 1440 | 1370 | | Polyneuritis |
| 10 | Red cock .. | 1485 | 1315 | 1200 | | Polyneuritis |
| 11 | Yellow and white cock .. | 1580 | 1580 | 1580 | | Polyneuritis |
| 12 | Black and yellow cock .. | 1630 | 1620 | 1370 | | Died Polyneuritis |

EXPERIMENT No. 26.—“Cargo” Rice.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | — |
|-----|----------------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|-------------------|
| 1 | Brown and yellow hen | 910 | 862 | 848 | 857 | 900 | 952 | 940 | Remains healthy. |
| 2 | Brown and black cock | 2005 | 2020 | 2085 | 2080 | 2062 | 2053 | 2052 | “ |
| 3 | Brown and red cock | 1805 | 1840 | 1885 | 1920 | 1965 | 1982 | 1982 | “ |
| 4 | Black hen | 1437 | 1450 | 1390 | 1290 | 1337 | 1235 | 1285 | Injured 3rd week. |
| 5 | Yellow hen | 887 | 868 | 745 | 835 | 980 | 1022 | 1080 | Remains healthy. |
| 6 | Brown cock | 1195 | 1265 | 1297 | 1325 | 1352 | 1375 | 1335 | “ |
| 7 | Brown hen | 1205 | 1290 | 1405 | 1465 | 1530 | 1565 | 1425 | “ |
| 8 | Brown cock | 1035 | 1275 | 1112 | 1158 | 1164 | 1185 | 1195 | “ |
| 9 | Brown hen | 1012 | 1120 | 1130 | 1245 | 1265 | 1235 | 1205 | “ |
| 10 | White hen | 805 | 857 | 840 | 820 | 798 | 785 | 775 | “ |
| 11 | Black hen | 1048 | 1075 | 1007 | 1132 | 1152 | 1185 | 1190 | “ |
| 12 | Brown hen | 978 | 1010 | 1018 | 1045 | 1103 | 1135 | 1210 | “ |

Experiment with Unpolished Rices.

During the course of the experiments it was observed that fowls always remained in health on padi and fowls suffering from polyneuritis almost invariably recovered when given padi. The present experiment was one in which a partially husked rice known commercially as "cargo rice" was employed—about half the grains are denuded of husk. This experiment was carried out as a preliminary to certain experiments in which this same product, after milling in various ways, was employed. These latter experiments will be referred to later.

In the experiment now under review, Experiment No. 26, twelve fowls were fed on partially husked rice for six weeks and all remained in good health at the conclusion of the experiment.

Other experiments in which padi and unpolished rices were employed are detailed elsewhere—all these experiments confirmed the observation that fowls remained in health when fed on rice which still retained its subpericarpal layers.

Experiments with Parboiled Rice.

Experiments with parboiled rice were carried out simultaneously with and for longer periods than experiments with white polished rice, as controls for the results obtained in the latter groups.

The experiment first described, No. 27, was one undertaken at the beginning of the series. In it five fowls only were employed. All maintained health and weight during fifteen weeks, cooked unpolished parboiled rice only being supplied throughout. It was thus shown that, in the conditions under which our experiments were carried out, a diet of parboiled rice

and water was sufficient to maintain fowls in health and weight over prolonged periods and this result repeatedly obtained was regarded as an adequate control to results obtained in shorter periods, five weeks or less.

In Experiment No. 28 that form of parboiled rice known as "muthu samba" which is prepared in India was employed. It is an expensive variety and is eaten only by the more wealthy members of the Indian community. Many fowls in this group showed a moderate gain in weight at the end of six weeks, a few showed a slight loss, all remained healthy throughout.

The next experiment of this series, No. 29, was used as a control to the results obtained in another group of fowls fed on the same sample of rice after exhaustion with proof spirit (in the latter group four cases of polyneuritis occurred within six weeks). In the experiment now under discussion, which was continued for ten weeks, though most of the fowls lost materially in weight and one died in the 9th week, none showed recognisable signs of polyneuritis.

In Experiment No. 30 parboiled rice in a finely ground state was used as a control to the results in groups in which a similar product after exhaustion with alcohol was employed.

It is apparent from an analysis of the results in this series of experiments that marked differences exist in the nutritive value of different samples of parboiled rice. These differences are due to two factors, the initial richness of the grain and the extent to which the polishing process has been carried out. It does not appear that the method of treatment by parboiling before husking operates in any way other than to harden the external layers of the grain rendering them less easy of removal.

EXPERIMENT No. 27.—Unpolished Parboiled Rice.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. | 8th
week. | 9th
week. | 10th
week. | 11th
week. | 12th
week. | 13th
week. | 14th
week. | 15th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|---------------|---------------|---------------|---------------|---------------|---------------|
| 1 | Black hen .. | 822 | 755 | 740 | 740 | 700 | 700 | 720 | 760 | 710 | 710 | 715 | 704 | 725 | 727 | 747 | 760 |
| 2 | Red hen .. | 727 | 785 | 770 | 770 | 740 | 740 | 730 | 740 | 740 | 750 | 745 | 765 | 785 | 790 | 825 | 850 |
| 3 | Black hen .. | 642 | 665 | 645 | 650 | 630 | 630 | 620 | 630 | 635 | 630 | 625 | 635 | 635 | 657 | 700 | 700 |
| 4 | Grey hen .. | 807 | 810 | 807 | 810 | 790 | 820 | 820 | 830 | 830 | 850 | 870 | 850 | 860 | 864 | 902 | 912 |
| 5 | Brown cock .. | 825 | 875 | 872 | 855 | 810 | 790 | 810 | 810 | 800 | 830 | 802 | 789 | 785 | 792 | 820 | 860 |

EXPERIMENT No. 28.—Parboiled Rice (Indian).

| No. | Description | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Brown hen .. | .. | 1300 | 1298 | 1292 | 1290 | 1270 | 1270 |
| 2 | Red cock .. | .. | 1180 | 1194 | 1185 | 1160 | 1175 | 1190 |
| 3 | Black cock .. | .. | 1192 | 1155 | 1152 | 1125 | 1140 | 1140 |
| 4 | Brown cock .. | .. | 1292 | 1322 | 1320 | 1277 | 1280 | 1275 |
| 5 | Red hen .. | .. | 1117 | 1130 | 1094 | 1075 | 1025 | 1045 |
| 6 | Black cock .. | .. | 1345 | 1339 | 1307 | 1305 | 1325 | 1310 |
| 7 | Brown hen .. | .. | 1212 | 1192 | 1185 | 1205 | 1210 | 1185 |
| 8 | Grey hen .. | .. | 1210 | 1190 | 1167 | 1187 | 1245 | 1255 |
| 9 | Brown cock .. | .. | 1660 | 1810 | 1850 | 1852 | 1835 | 1820 |
| 10 | Black hen .. | .. | 1280 | 1340 | 1307 | 1245 | 1200 | 1190 |
| 11 | Black hen .. | .. | 1272 | 1335 | 1295 | 1285 | 1280 | 1300 |
| 12 | Brown hen .. | .. | 1350 | 1395 | 1425 | 1484 | 1550 | 1410 |

| Protein. | Fat. | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 6.3 | 0.2 | 78.31 | 14.35 | 0.84 |
| | | P ₂ O ₅ | | |
| | | 0.345 | | |

EXPERIMENT No. 29.—Parboiled Rice (Penang).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. | 8th
week. | 9th
week. | 10th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|-------------------------|
| 1 | Yellow hen .. | .. | 1315 | 1340 | 1310 | 1200 | 1155 | 1125 | 1100 | 1065 | 1040 | 972 |
| 2 | White cock .. | .. | 1300 | 1285 | 1240 | 1205 | 1155 | 1135 | 1120 | 1050 | 1005 | 1045 |
| 3 | Yellow hen .. | .. | 920 | 900 | 905 | 915 | 839 | 820 | 810 | 802 | 840 | 775 |
| 4 | Red cock .. | .. | 1390 | 1435 | 1450 | 1470 | 1435 | 1380 | 1405 | 1420 | 1445 | 1398 |
| 5 | Yellow hen .. | .. | 1270 | 1220 | 1140 | 1055 | 1005 | 1005 | 955 | 885 | 825 | 695 |
| 6 | Black cock .. | .. | 1380 | 1385 | 1370 | 1325 | 1310 | 1310 | 1295 | 1272 | 1275 | 1235 |
| 7 | Yellow hen .. | .. | 1345 | 1370 | 1385 | 1380 | 1295 | 1270 | 1240 | 1082 | 955 | Died. No Polyn neuritis |
| 8 | Yellow cock .. | .. | 1700 | 1700 | 1565 | 1565 | 1547 | 1545 | 1505 | 1515 | 1500 | 1505 |
| 9 | Brown hen .. | .. | 1450 | 1355 | 1350 | 1335 | 1220 | 1205 | 1200 | 1109 | 1098 | 885 |
| 10 | Yellow cock .. | .. | 1475 | 1465 | 1410 | 1385 | 1420 | 1410 | 1355 | 1395 | 1400 | 1380 |
| 11 | Yellow hen .. | .. | 1195 | 1205 | 1190 | 1130 | 1122 | 1090 | 1160 | 1084 | 1040 | 1030 |
| 12 | Black cock .. | .. | 1590 | 1600 | 1585 | 1540 | 1510 | 1470 | 1480 | 1472 | 1458 | 1455 |

| Protein. | Fat | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 7.80 | 0.50 | 76.62 | 14.30 | 0.78 |
| | | P ₂ O ₅ | | |
| | | 0.41 | | |

EXPERIMENT No. 30.—Parboiled Rice (Penang) Finely Ground.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Black hen .. | .. | 1210 | 1225 | 1180 | 1285 | 1280 | 1245 |
| 2 | White hen .. | .. | 1320 | 1275 | 1239 | 1265 | 1255 | 1272 |
| 3 | Grey hen .. | .. | 1450 | 1430 | 1337 | 1385 | 1405 | 1352 |
| 4 | Brownish hen | .. | 1235 | 1205 | 1187 | 1172 | 1140 | 1150 |
| 5 | Brown cock .. | .. | 1185 | 1195 | 1157 | 1170 | 1150 | 1079 |
| 6 | Brown hen .. | .. | 1225 | 1240 | 1246 | 1277 | 1280 | 1220 |
| 7 | White hen .. | .. | 1305 | 1280 | 1238 | 1257 | 1265 | 1275 |
| 8 | Black hen .. | .. | 1185 | 1190 | 1191 | 1245 | 1237 | 1202 |
| 9 | Brown hen .. | .. | 1240 | 1165 | 1055 | 1072 | 1102 | 1100 |
| 10 | Black hen .. | .. | 1330 | 1290 | 1173 | 1195 | 1177 | 1170 |
| 11 | White hen .. | .. | 1305 | 1295 | 1219 | 1280 | 1235 | 1195 |
| 12 | Brown cock .. | .. | 1375 | 1335 | 1304 | 1300 | 1250 | 1222 |

Experiments with Malay Rice.

It is conceded by all those whose knowledge of this country and of its people enable them to speak with authority upon the point that among Malays under primitive conditions beri-beri is very rare.

Braddon states that "among these natives so long as they lead their primitive pastoral and agricultural life untouched by the influences which march with a civilization represented by encroaching hordes of Chinese beri-beri never occurs."

Hamilton Wright (16) says "My own experience of Malay Kampongs (Malay villages) is that beri-beri is almost unknown in them. The farther the Malay population is removed from centres of civilization the less beri-beri is seen in it."

Daniels (17) says "Malays living in Kampongs are the only class that do not suffer from beri-beri."

An account has already been given of the Malay method of preparing rice.

The first experiment in this series (Experiment No. 31) was one in which a rice prepared from a locally grown padi was employed. This was obtained from the Kuala Pilah district through the kindness of the District Officer, Mr. Eric Dickson, and Dr. Lucy.

A Malay woman prepared rice from this padi after the manner and with the primitive implements used by Malays in their own villages.

As will be seen from the table, of the twelve fowls employed for this experiment all remained healthy with the exception of No. 12 which on the 42nd day of the experiment developed polyneuritis. The attack ran the usual course and on emulsion of rice polishings and padi the fowl recovered completely in 6 weeks.

In the second experiment in this series (Experiment No. 32) which was one of a group of experiments to which particular attention will be directed later Malay rice was prepared from a partially husked padi imported from Indo-China.

The method of preparation of this rice was similar to that employed in Experiment No. 31.

As the supply of this product was limited only eight fowls were employed for this experiment and it lasted only five weeks. All the fowls remained normal throughout this period.

The occurrence of a case of polyneuritis in a fowl fed on "Malay" rice calls for some comment. It is the single instance throughout these experiments in which hundreds of fowls have been employed, in which polyneuritis developed in a fowl on a diet other than white polished rice. The clinical appearances were in all respects identical with those of other cases of polyneuritis and we do not assign to the case an origin other than dietary. It appears that even the limited amount of polishing to which Malay rice is subjected in its preparation may on occasion be harmful.

EXPERIMENT No. 31.—Malay Rice prepared from Malay padi.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | — |
|-----|----------------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|---------------------------------|
| 1 | Red cock | .. | 1542 | 1355 | 1465 | 1332 | 1440 | 1315 | |
| 2 | Brown hen | .. | 1282 | 1487 | 1425 | 1443 | 1430 | 1440 | |
| 3 | Red cock | .. | 1348 | 1295 | 1250 | 1272 | 1300 | 1264 | |
| 4 | Black and white hen | .. | 1145 | 1270 | 1210 | 1282 | 1215 | 1160 | |
| 5 | Brown hen | .. | 1182 | 1185 | 1185 | 1162 | 1240 | 1260 | |
| 6 | Yellow and black hen | .. | 902 | 933 | 948 | 907 | 828 | 837 | |
| 7 | Black hen | .. | 1225 | 1262 | 1295 | 1317 | 1335 | 1352 | |
| 8 | Black hen | .. | 1130 | 1125 | 1180 | 1185 | 1140 | 1170 | |
| 9 | Black hen | .. | 1145 | 1295 | 1148 | 1230 | 1235 | 1262 | |
| 10 | Black hen | .. | 1302 | 1235 | 1275 | 1265 | 1245 | 1260 | |
| 11 | Red cock | .. | 1180 | 1170 | 1172 | 1190 | 1142 | 1202 | |
| 12 | Brown hen | .. | 1140 | 1093 | 1085 | 885 | 780 | 678 | Developed polynuritis 7th week. |

| Protein. | Fat. | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 7.2 | 0.63 | 77.29 | 14. | 0.88 |
| | | P ₂ O ₅ | | |
| | | 0.37 | | |

EXPERIMENT No. 32.—Malay Rice prepared from "Cargo Rice."

| No | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | — |
|----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|---------------------|
| 1 | Yellow hen .. | 1045 | 1062 | 1092 | 1070 | 1090 | 1095 | Healthy throughout. |
| 2 | Yellow cock .. | 1685 | 1685 | 1705 | 1680 | 1695 | 1700 | " " |
| 3 | Brown hen .. | 1425 | 1409 | 1383 | 1357 | 1367 | 1385 | " " |
| 4 | Brown hen .. | 1525 | 1676 | 1675 | 1525 | 1475 | 1505 | " " |
| 5 | Brown cock .. | 1660 | 1730 | 1740 | 1710 | 1690 | 1720 | " " |
| 6 | White hen .. | 775 | 769 | 730 | 735 | 748 | 755 | " " |
| 7 | Brown hen .. | 940 | 980 | 965 | 917 | 942 | 990 | " " |
| 8 | Brown hen .. | 1210 | 1225 | 1300 | 1327 | 1330 | 1405 | " " |

| Protein. | Fat. | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 7.7 | 0.23 | 75.43 | 15.5 | 1.14 |
| | | P ₂ O ₆ | | |
| | | 0.52 | | |

Experiments with Polishings.

As it now appeared that the harmfulness of a rice diet varied directly as the extent to which the rice had been milled, it was determined to test this hypothesis further by adding to a white polished rice, which when given alone had been shown to be harmful, a quantity of polishings equal in amount to that which had been milled away. The miller estimates that from 40 parts of padi there are produced 25 parts of rice and 5 parts of polishings; 16% of the husked grain is thus lost in polishing. An actual weighing of some thousands of grains of unpolished and polished rice from the same lot of padi showed that the loss was about 10%.

In the first experiment of this series, Experiment No. 33, the white polished rice selected was the same sample as that employed in Experiment No. 15, and which when fed alone had resulted in six cases of polyneuritis among twelve fowls in five weeks.

The rice polishings were from the same sample. They were sifted in order to remove husks and broken rice and given in the form of an emulsion with distilled water.

During the first and second weeks of the experiment an amount of emulsion equal to 10 grammes of polishings was given daily for a fowl eating 60 grammes of rice.

During the 3rd week 8 grammes of polishings were given daily, during the 4th and 5th weeks 6 grammes, during the 6th week 4 grammes and during the 7th week 3 grammes were given.

Up to the 7th week all the fowls gained slightly in weight. In the 7th week there was a moderate loss amounting to 2.6 grammes per kilogramme of body weight.

In the 7th week there was a moderate loss 2.6 grammes per kilo. It was concluded therefore that the amount of polishings necessary to add to 60 grammes of this sample of white polished rice to maintain the normal nutritive equilibrium was between 3 and 4 grammes, say 3.5 grammes. This amount being added there was in the eighth week a slight gain in weight in the whole group.

It was considered desirable to repeat this result with a rice from a beri-beri outbreak. An experiment was therefore planned in which eight fowls were fed on the Durian Tipus rice, each alternate fowl receiving in addition an amount of polishings equal to 3.5 grammes per kilo of body weight.

The result is as shown by the Experiment No. 34. In four weeks two of the fowls fed on rice alone had developed polyneuritis and all four had lost weight while the four fowls which were given polishings in addition remained healthy and at the conclusion of the experiment all had gained in weight.

The conclusion was now arrived at that certain essential substances are lacking in the case of white polished rice and that the addition of rice polishings to a diet of white polished rice prevents the occurrence of polyneuritis in fowls.

EXPERIMENT No. 33.—White Polished Rice + Polishings.

| No. | Description. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. | 6th week. | 7th week. | 8th week. |
|-----|----------------|--------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| 1 | Yellow cock .. | .. | 1402 | 1375 | 1362 | 1385 | 1365 | 1320 | 1235 | 1240 |
| 2 | Black hen .. | .. | 655 | 655 | 648 | 652 | 635 | 635 | 620 | 580 |
| 3 | Red hen .. | .. | 870 | 890 | 902 | 925 | 980 | 1005 | 1010 | 1030 |
| 4 | Brown hen .. | .. | 672 | 740 | 745 | 745 | 737 | 735 | 700 | 692 |
| 5 | White hen .. | .. | 693 | 770 | 780 | 792 | 770 | 782 | 735 | 717 |
| 6 | Yellow hen .. | .. | 1262 | 1345 | 1390 | 1400 | 1420 | 1420 | 1352 | 1372 |
| 7 | Grey hen .. | .. | 752 | 805 | 818 | 772 | 815 | 818 | 795 | 770 |
| 8 | Brown hen .. | .. | 1648 | 1620 | 1612 | 1615 | 1590 | 1605 | 1525 | 1585 |
| 9 | Yellow cock .. | .. | 2015 | 2154 | 2170 | 2193 | 2137 | 2104 | 2095 | 2044 |
| 10 | Red cock .. | .. | 1437 | 1510 | 1518 | 1525 | 1540 | 1548 | 1550 | 1580 |
| 11 | White cock .. | .. | 1370 | 1372 | 1373 | 1387 | 1378 | 1398 | 1385 | 1440 |
| 12 | Red cock .. | .. | 1355 | 1440 | 1350 | 1462 | 1505 | 1525 | 1540 | 1565 |

| | Protein. | Fat. | Carbohydrate. | Moisture. | Ash. | P ₂ O ₅ |
|---------------------|----------|------|---------------|-----------|------|-------------------------------|
| White polished rice | 7.3 | 0.40 | 77.43 | 14.3 | 0.57 | 0.28 |
| Polishings | 11.7 | 10.0 | 59.05 | 11.95 | 7.3 | 4.6 |

EXPERIMENT No. 34.—Nos. 1, 3, 5, 7 White Polished Rice +
Polishings Nos. 2, 4, 6, 8, White Rice only.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Black hen .. | 1237 | 1309 | 1380 | 1398 | 1383 |
| 2 | Red cock .. | 1254 | 1200 | 1130 | 1047 | Polyneuritis |
| 3 | White cock .. | 1625 | 1615 | 1625 | 1635 | 1630 |
| 4 | Brown hen .. | 890 | 875 | 820 | 827 | 778 |
| 5 | White hen .. | 875 | 865 | 900 | 900 | 900 |
| 6 | White cock .. | 1810 | 1648 | 1460 | 1162 | Polyneuritis |
| 7 | Yellow hen .. | 918 | 970 | 991 | 1030 | 1040 |
| 8 | Brown hen .. | 1005 | 965 | 895 | 817 | 820 |

| | Protein. | Fat. | Carbohydrate. | Moisture. | Ash. | P ₂ O ₅ |
|-------------------|----------|------|---------------|-----------|------|-------------------------------|
| White Rice (D.T.) | 7.45 | 0.17 | 78.02 | 13.85 | 0.51 | 0.28 |
| Polishings | 11.7 | 10.0 | 59.05 | 11.95 | 7.3 | 4.6 |

The Phosphorous Content of Rice as an Indicator of the extent to which rice has been polished.

Having proved that the occurrence of polyneuritis in fowls and of beri-beri in man is due to the consumption of rice from which the subpericarpal layers have been removed by the process of polishing, that such rice is equally harmful whether freshly prepared or stored for months, the development in it of a poison is excluded and it is necessary to revert to the consideration of the results obtained from analyses of rices.

In the tables the results of these analyses are given both for the undried and dried materials. The table showing the results calculated on the dried materials facilitates comparison.

Comparing the diet unpolished rice with that of polished rice it will be seen that there are considerable differences. A diet of polished rice made up with polishings to the necessary amount for the maintenance of weight and health approximates in composition to one of unpolished rice, and it might therefore be inferred that the deficiency of white rice in respect of proteins, fats, and salts was accountable for its harmful results. The protein content of polished rice differs only by a slight amount from that of two parboiled rices which were proved harmless and allowing for experimental errors the differences are apparently insufficient to account for the results. In respect of fat the polished rice is poorer but the difference is a small one and, as has been previously stated, can hardly account for the difference in the results. The ash gives a very imperfect representation of the salts present but there is a considerable difference in the amount of ash present in the unpolished and polished rices yet even the amount present in the latter rice exceeds that obtained from one of the parboiled rices.

Now in the case of parboiled rice extracted with hot alcohol (94%) the amount of fat removed is relatively great but of proteins and ash relatively small and in the case of parboiled rice extracted with proof spirit similar results are observed.

The proteins were invariably estimated by the Kjeldahl process and allowing for the possibilities of experimental error it must be admitted that the differences in respect of proteins as determined by this method will not explain the results.

The question of the carbohydrates cannot seriously be considered as these were calculated by difference.

These methods of analysis failed in every way to explain the results and it was necessary to employ other methods. It was suggested to one of us by Dr. Mott that the deficiency of white rice in lipoids might be of importance and various attempts were made to determine the amount of these present in rices but the process was a tedious one and the results not satisfactory. It was decided therefore to estimate the Phosphorus Pentoxide content of the various rices.

ESTIMATION OF PHOSPHORUS PENTOXIDE (P_2O_5)

Two methods were employed for the conversion of the phosphorus to inorganic combinations.

By the first process—

1. A weighed quantity of the material was burned in a platinum basin and the complete combustion of the organic matter was facilitated by sprinkling pure Potassium Nitrate over the ash.

2. The residue was treated with 10 c.c. of strong Hydrochloric Acid and evaporated to dryness over a water bath.

3. The residue was heated with 5 c.c. strong Hydrochloric Acid and 50 c.c. of distilled water for five minutes.

4. Filtered and the filter-paper washed with hot water till the washings ceased to give a precipitate with Silver Nitrate and Nitric Acid.

5. The combined filtrate and washings were evaporated to about 50 c.cm.

By the second process—

1. A weighed quantity of the material was placed in a Kjeldahl flask and heated with concentrated Sulphuric Acid and Potassium Hydrogen Sulphate until the fluid became colourless.

2. The fluid was diluted with water and filtered. The filter-paper was washed with water until the washings ceased to give a precipitate with Barium Chloride.

3. The combined filtrate and washings were evaporated to about 50 c.cm.

4. The concentrated filtrate and washings obtained either by the first process or the second process were neutralized with Ammonium Hydrate and a drop or two of Nitric Acid added to clear.

5. To the solution 10 grammes of Ammonium Nitrate were added, the solution warmed to about 40°C and excess of Molybdic solution added.

6. The mixture was kept in a warm place overnight and in the morning filtered. The precipitate was washed with a cold solution of Ammonium Nitrate (5%).

7. The Phospho-molybdate precipitate was dissolved in hot water and Ammonium Hydrate, it was so arranged

that the filtrate and washings measured 100 c.c. or thereabouts.

8. The solution of Phospho-molybdate was neutralized with Hydrochloric Acid and excess of Magnesia Mixture added. After half an hour 30 c.c. of Ammonium Hydrate (10%) were added.

9. The mixture was allowed to stand overnight. In the morning it was filtered and the precipitate washed with Ammonium Hydrate (2.5%) till free from chloride.

10. The precipitate was dried, ignited and the residue of Magnesium Pyrophosphate ($Mg_2 P_2O_7$) weighed. From this weight the amount of Phosphorus Pentoxide was calculated by the use of the factor 0.6394.

By whichever method the conversion of the phosphorous into inorganic combination is effected the results obtained are the same and it is entirely a matter of individual choice which shall be selected.

The gravimetric process was employed in preference to the volumetric because of its greater accuracy and now that the facts have been determined there is no reason why the volumetric process should not be employed.

Nearly two hundred estimations have been made and the results were invariably recorded on the undried material. The percentage of moisture in various kinds of rice does not vary greatly, being usually from 13—14% or thereabouts, and it was found that the percentage calculated on the dried material did not yield any information other than that obtained from that calculated on the undried material.

Polished white rice of the kind commercially known as Siam rice yields on an average 0.26% of Phosphorus Pentoxide and

is the variety usually associated with severe outbreaks of beri-beri. Schaumann gives an analysis of a rice yielding only 0.1% Phosphorous Pentoxide. This is lower than any result obtained here.

Polished white rice of the kind commercially known as Rangoon rice yields on an average 0.328% of Phosphorus Pentoxide and the incidence of beri-beri is less on this kind of rice than the former.

Malay rice yields on an average 0.38% of Phosphorus Pentoxide and the incidence of beri-beri is still less on this kind of rice.

Parboiled rice yields on an average 0.415% or over of Phosphorus Pentoxide and beri-beri does not occur when this kind of rice is eaten.

Unpolished rice yields on an average 0.54% of Phosphorus Pentoxide and can never produce beri-beri.

The great increase in the consumption of parboiled rice has induced the local millers to improve the appearance of their product and this result some of them have sought to achieve by polishing or as it is technically called "pearling." Attention has previously been directed to the difficulty of accomplishing this on account of the toughening of the grain by soaking and steaming but by the use of stone polishers it is possible to remove a considerable amount of the subpericarpal layers with consequent diminution of the Phosphorus Pentoxide content. We recently examined such a rice which only yielded 0.34% of Phosphorus Pentoxide (see Experiment 30).

Another sample of parboiled rice after hulling was examined and yielded 0.6% of Phosphorus Pentoxide. After pearling once it yielded 0.5% and after pearling twice it yielded 0.4%.

An unpolished rice yielding 0.56% of Phosphorus Pentoxide was polished in the ordinary way and the polished or white rice yielded only 0.26%.

AVERAGE RESULTS OF ALL ESTIMATIONS.

| | | | | Percentage of |
|-------------------------|-----|-----|-----|---------------|
| | | | | P_2O_5 |
| Polished rice (Siam) | ... | ... | ... | 0.26 |
| Polished rice (Rangoon) | ... | ... | ... | 0.328 |
| Malay rice | ... | ... | ... | 0.38 |
| Parboiled rice | ... | ... | ... | 0.415 |
| Unpolished rice | ... | ... | ... | 0.54 |

Fowls fed on polished rice and receiving polishings in sufficient amount are receiving a dietary which approximates in its content of Phosphorus Pentoxide to a diet of unpolished rice. The harmfulness of rice is therefore in inverse proportion to its Phosphorus Pentoxide content and in direct proportion to the extent to which it has been polished.

None of the rices connected with outbreaks of beri-beri yielded more than 0.26% of Phosphorous Pentoxide. The rices substituted for these and which were effective in preventing the continuance of the outbreaks yielded not less than 0.4% of that substance.

Attention should be directed to the percentage of the Phosphorus Pentoxide in parboiled rice after extraction with 94% alcohol, the diminution in amount is extremely small and apparently negatives the vital importance of this substance. In practice extracted rice would never be under consideration and the value of this estimation as an indicator of the extent to which rice has been polished has stood the test of numerous experiments.

It will be contended that the estimation of any of the other constituents of rice would serve equally well for this purpose. It has already been shown that the estimation of

proteins is not satisfactory. The removal of the total fats from a rice is an extremely tedious process and the determination of total ash is complicated with errors of volatilization and incomplete ashing.

Admitting the value of those other estimations it must still be conceded that the estimation of Phosphorus Pentoxide permits of a reasonable margin of error and furnishes differences which can be accurately determined and are more striking than would be furnished by any of the other constituents.

ANALYSIS OF RICES.

| | | | Protein. | Fat. | Carbo-
hydrates. | Ash. | Moisture. | Percentage
of Phosphorus
pentoxide. | Effect on fowls:
estimated by
the occurrence
of polyneuritis. |
|----|--|-------|----------|-------|---------------------|------|-----------|---|--|
| 1 | Polishings | | 13.7 | 14.16 | 52.77 | 7.54 | 11.83 | 4.1 | — |
| 2 | Unpolished rice .. | | 9.0 | 1.65 | 75.52 | 1.08 | 12.75 | 0.56 | + |
| 3 | Polished rice .. | | 8.6 | 0.22 | 76.23 | 0.6 | 14.35 | 0.76 | + |
| 4 | Polished rice (washed) | | 6.2 | 0.22 | 75.04 | 0.34 | 16.2 | 0.21 | + |
| 5 | Polished rice (washed) plus polishings | | 8.61 | 1.29 | 73.35 | 0.89 | 15.86 | 0.463 | — |
| 6 | Parboiled rice .. | | 7.55 | 0.45 | 77.76 | 0.94 | 13.3 | 0.427 | — |
| 7 | The same rice after extraction with 94% alcohol | | 7.6 | 0.06 | 80.66 | 0.88 | 10.8 | 0.425 | + |
| 8 | Parboiled rice .. | | 7.8 | 0.5 | 76.62 | 0.78 | 14.3 | 0.41 | — |
| 9 | The same rice after extraction with proof spirit | | 7.65 | 0.10 | 80.01 | 0.44 | 11.8 | 0.39 | + |
| 10 | Siam rice (Depot) | | 7.8 | 0.15 | 77.49 | 0.56 | 14 | 0.28 | + |
| 1 | Siam rice (D. T.) | | 7.45 | 0.17 | 78.02 | 0.51 | 13.85 | 0.28 | + |
| 2 | Rangoon rice .. | | 7.3 | 0.63 | 77.15 | 0.74 | 14.18 | 0.33 | + |
| 3 | Indian rice .. | | 6.3 | 0.2 | 78.31 | 0.84 | 14.35 | 0.345 | — |
| 4 | Malay rice from cargo rice | | 7.7 | 0.23 | 75.43 | 1.14 | 15.5 | 0.51 | — |
| 5 | Malay rice from Malay padi | | 7.2 | 0.63 | 77.29 | 0.88 | 14.0 | 0.37 | + |
| 6 | Siam rice No. 2 quality stale | | 7.3 | 0.40 | 77.43 | 0.57 | 14.3 | 0.28 | + |
| 7 | Siam rice No. 2 quality freshly milled | | 8.1 | 0.41 | 76.76 | 0.47 | 14.26 | 0.27 | + |
| 8 | Siam rice No. 1 quality .. | | 6.9 | 0.20 | 77.26 | 0.40 | 15.24 | 0.22 | + |

ANALYSIS OF RICES (CALCULATIONS BASED ON DRIED MATERIALS).

| | | | Protein. | Fat. | Carbo-
hydrates. | Ash. | Percentage of
Phosphorus
Pentoxide. | Effect on
fowl:
estimated by
the occurrence
of polyneuritis. |
|-----|--|----|----------|------|---------------------|------|---|--|
| 1a. | Polishings | .. | 15.5 | 16.0 | 65.0 | 8.5 | 4.6 | |
| 2a. | Unpolished rice | .. | 10.3 | 1.89 | 86.58 | 1.23 | 0.64 | — |
| 3a. | Polished rice | .. | 10.0 | 0.25 | 89.05 | 0.7 | 0.3 | + |
| 4a. | Polished rice (washed) | .. | 9.7 | 0.26 | 89.64 | 0.4 | 0.25 | + |
| 5a. | Polished rice (washed) plus polishings | .. | 10.2 | 1.33 | 87.22 | 1.05 | 0.55 | — |
| 6a. | Parboiled rice | .. | 8.7 | 0.51 | 89.79 | 1.0 | 0.492 | — |
| 7a. | The same rice after extraction with 94% alcohol | .. | 8.5 | 0.06 | 90.46 | 0.98 | 0.476 | + |
| 8a. | Parboiled rice | .. | 9.1 | 0.58 | 89.76 | 0.56 | 0.47 | — |
| 9a. | The same rice after extraction with proof spirit | .. | 8.6 | 0.11 | 90.79 | 0.5 | 0.32 | + |

The results of numerous experiments had now made it clear that the addition of rice polishings to a diet of white polished rice prevented the harmful effects of such a diet. The further researches consisted in an endeavour to determine the nature of the substances in rice polishings which were responsible for this result.

In order that the results in the various experiments of the series should be comparable it was considered desirable that the various rices and rice products employed should be derived from the same lot of padi. Accordingly we obtained from a rice mill in Singapore the materials desired:—

(1) A quantity of the rice as it passed from the huller to the polishing machines. At this stage the grain is deprived of the husk only (unpolished rice).

(2) A quantity of the rice after having passed through the polishing machines (white polished rice).

(3) A quantity of the rice polishings or rice meal collected from the polishing machines.

A preliminary series of experiments was carried out to test the value of these various foodstuffs when fed to fowls.

In Experiment No. 35 the unpolished rice was employed. It was shown that this foodstuff in addition to which only water was supplied sufficed to maintain fowls in good health for many weeks.

In Experiment No. 36 the white polished rice was employed. Within three weeks six fowls out of a total of twelve fowls included in the experiment suffered from polyneuritis.

In Experiment No. 37 the white polished rice diet identical with that in Experiment No. 36 was employed. In addition each fowl received daily 5 grammes of sifted rice polishings emulsified in water. All the fowls remained healthy.

This series of experiments confirmed results previously obtained and showed that these materials were suitable for employment in the further researches which it was proposed to undertake.

As an instance of the practical application of these principles, Dr. J. M. Atkinson, Principal Civil Medical Officer, Hongkong permits us to refer to some notes he has made in regard to the medical history of the Victoria Gaol, Hongkong. At the meeting of the Far Eastern Association of Tropical Medicine at Manila in March 1909 we communicated the results of our observations in regard to the causation of beri-beri and advanced the view that the use of unpolished rice would be found an effective preventive measure. Dr. Atkinson who was the official representative of Hongkong at the meeting, on his return to the Colony made inquiries as to the diets in the various public institutions. He found that in the Victoria Gaol which had been singularly free from beri-beri, unpolished rice had been in use for many years. The average daily number of prisoners in this Gaol is over 500, many of them being long sentence prisoners.

The Chief Superintendent of Police reported the facts as follows.

“No case of beri-beri has originated in the jail in the last fifteen years. Polished rice is not supplied to the prisoners and never has been. They have always been given unpolished rice as it is cheaper than polished rice.”

As beri-beri is one of the principal diseases afflicting the native population of Hongkong this observation is of great

importance as showing the value of an unpolished rice diet in preventing beri-beri.

M. Breaudat and Dr. Denier of the Pasteur Institute Saigon have recently published the report of an experiment, the results of which showed the value of rice polishings (*son*) as a preventive of human beri-beri.

EXPERIMENT No. 35.—Unpolished Rice.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | 6th
week. | 7th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | 1380 | 1380 | 1380 | 1370 | 1380 | 1370 | 1395 | 1420 |
| 2 | Red cock .. | 1145 | 1195 | 1205 | 1205 | 1220 | 1255 | 1280 | 1310 |
| 3 | Black cock .. | 1080 | 1115 | 1170 | 1180 | 1180 | 1125 | 1160 | 1180 |
| 4 | Yellow hen .. | 1035 | 1090 | 1100 | 1072 | 1070 | 1040 | 1020 | 1040 |
| 5 | Black cock .. | 1145 | 1200 | 1280 | 1305 | 1370 | 1345 | 1320 | 1340 |
| 6 | Yellow hen .. | 1450 | 1460 | 1330 | 1305 | 1345 | 1270 | 1275 | 1240 |
| 7 | Black hen .. | 925 | 920 | 930 | 935 | 925 | 940 | 960 | 940 |
| 8 | Yellow cock .. | 1185 | 1105 | 1175 | 1265 | 1330 | 1300 | 1325 | 1285 |
| 9 | Brown hen .. | 1640 | 1660 | 1430 | 1350 | 1360 | 1415 | 1390 | 1380 |
| 10 | Red cock .. | 1405 | 1450 | 1450 | 1475 | 1475 | 1340 | 1390 | 1430 |
| 11 | Yellow hen .. | 1030 | 980 | 990 | 940 | 995 | 980 | 980 | 995 |
| 12 | Brown cock .. | 1020 | 1060 | 1060 | 1035 | 1030 | 1035 | 950 | 1050 |

| Protein. | Fat. | Carbohydrate. | Moisture. | Ash. |
|----------|------|-------------------------------|-----------|------|
| 9.0 | 1.65 | 75.52 | 12.75 | 1.08 |
| | | P ₂ O ₅ | | |
| | | 0.56 | | |

EXPERIMENT No. 36.—White polished Rice (washed).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|
| 1 | Black cock .. | 1175 | 1045 | 942 | 810 |
| 2 | Black hen .. | 1205 | 1115 | 1082 | Polyneuritis |
| 3 | Red cock .. | 1300 | 1250 | 1165 | Polyneuritis |
| 4 | Brown hen .. | 1595 | 1470 | 1370 | 1255 |
| 5 | Yellow hen .. | 1390 | 1210 | 1098 | 985 |
| 6 | Black hen .. | 1025 | 990 | 935 | 795 |
| 7 | White hen .. | 1180 | 1175 | 1060 | 940 |
| 8 | Yellow hen .. | 1470 | 1395 | 1275 | 1120 |
| 9 | Black hen .. | 1210 | 1150 | 1115 | Polyneuritis |
| 10 | Brown hen .. | 1370 | 1190 | 1025 | Polyneuritis |
| 11 | Red cock .. | 1950 | 1810 | 1635 | Polyneuritis |
| 12 | White cock .. | 1790 | 1720 | 1670 | Polyneuritis |

Protein. 8.2 Fat. 0.22 Carbohydrate. 75.04 Moisture. 16.2 Ash. 0.34

P₂O₅
0.21

EXPERIMENT No. 37.
White Polished Rice (washed) + Rice Polishings.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Brown cock .. | .. | 1390 | 1470 | 1490 | 1560 | 1575 |
| 2 | White hen .. | .. | 1470 | 1560 | 1470 | 1400 | 1430 |
| 3 | Black hen .. | .. | 1340 | 1410 | 1420 | 1400 | 1385 |
| 4 | Red cock .. | .. | 1505 | 1505 | 1510 | 1525 | 1505 |
| 5 | White hen .. | .. | 1720 | 1730 | 1630 | 1605 | 1575 |
| 6 | Brown hen .. | .. | 1215 | 1275 | 1300 | 1445 | 1325 |
| 7 | Black hen .. | .. | 1275 | 1350 | 1380 | 1340 | 1390 |
| 8 | Red cock .. | .. | 1925 | 1990 | 1955 | 1945 | 1930 |
| 9 | Black hen .. | .. | 1250 | 1230 | 1200 | 1150 | 1100 |
| 10 | White hen .. | .. | 1580 | 1530 | 1470 | 1535 | 1530 |

| | Protein. | Fat. | Carbohydrate. | Ash. | Moisture. | P ₂ O ₅ |
|----------------------------|----------|-------|---------------|------|-----------|-------------------------------|
| White polished Rice washed | 8.2 | .22 | 75.04 | 0.34 | 16.2 | .21 |
| Polishings | 13.7 | 14.16 | 52.77 | 7.54 | 11.83 | 3.55 |

The Effect of Exposure to High Temperatures.

At this point the various methods of analysis suitable for the recognition of the physiologically active substances were reviewed. The methods hitherto employed for the recognition of specific differences among the various rices and rice products experimented with had always involved the exposure of these substances to high temperatures.

Bearing in mind the results obtained by Grijns in his experiments with kachang idju (*Geneeskundig Tijdschrift voor Nederlandsch Indie* 1901) and by Holst and Fröhlich (18) with various meats and vegetables, it was decided to test the effect of exposure to high temperatures on the materials with which we were working.

The first substance experimented with was the rice polishings. Quantities of polishings sufficient for six fowls for one day were mixed with water in flasks and heated in the autoclave for one hour at 120°C. In Experiment No. 38 a group of fowls on a white polished rice diet were each given daily a portion of the emulsion so prepared equivalent to five grammes of polishings. No cases of polyneuritis appeared among them in the five weeks during which the experiment was in progress.

In the next experiment, No. 39, polishings heated for two hours at 120°C were employed. One fowl died in the third week of the experiment without showing the clinical signs of polyneuritis or the characteristic nerve changes on post-mortem examination.

This unexpected result caused us to initiate further experiments to determine whether physical conditions apart from the temperature were responsible for this result.

In Experiment No. 40, padi which had been sterilized in a small bag suspended in the wire basket of the autoclave for one hour at 120°C was employed. One group of fowls Nos. 1 to 6 were fed on this and another group Nos. 7 to 12 were fed on the untreated padi. This experiment gave a result identical with that of other observers.

In Experiment No. 41, unpolished rice was employed. Nos. 1 to 4 were fed on unpolished rice untreated. Nos. 5 to 8 were fed on unpolished rice sterilised in a porcelain basin in the autoclave for one hour at 120°C . Nos. 9 to 12 were fed on unpolished rice sterilised for two hours in the same way. The results of these experiments showed that when sterilised in an atmosphere of steam for one or two hours at 120°C the physiological activity of the protective substances was destroyed.

In Experiment No. 42, padi immersed in water in a porcelain basin and sterilised in a autoclave for one hour at 120°C was employed. The fowls remained healthy.

In Experiment No. 43 padi sterilised for one hour at 120°C in a hot-air steriliser was employed. The fowls remained healthy.

In certain large institutions rice is cooked by steam under pressure. By this method larger quantities are dealt with than by cooking in open vessels and the cooking is presumably more quickly carried out. An opportunity of making a practical test of the relative merits of these two methods of cooking we owe to the courtesy of Dr. Gray and Dr. Freer.

In Experiment No. 44 a group of fowls was fed on parboiled rice cooked in the usual way in open vessels. All the fowls remained healthy.

In Experiment No. 45 the same rice cooked by steam under pressure as issued to the inmates of a large institution

EXPERIMENT No. 38.—White Polished Rice + Polishings
in emulsion heated at 120°C for one hour.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | White cock .. | 1330 | 1400 | 1445 | 1463 | 1455 | 1485 |
| 2 | Brown cock .. | 1200 | 1245 | 1260 | 1220 | 1220 | 1195 |
| 3 | Black cock .. | 1345 | 1380 | 1375 | 1405 | 1415 | 1400 |
| 4 | Yellow cock .. | 1265 | 1170 | 1120 | 1180 | 1160 | 1065 |
| 5 | Black cock .. | 1420 | 1400 | 1515 | 1495 | 1515 | 1510 |
| 6 | Black cock .. | 1535 | 1515 | 1535 | 1510 | 1465 | 1475 |

EXPERIMENT No. 39.
White Polished Rice + Polishings in emulsion heated at 120°C for two hours.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. | — |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|----------------------------|
| 1 | White cock .. | 1485 | 1430 | 1340 | 1370 | 1380 | 1340 | |
| 2 | Brown cock .. | 1195 | 1175 | 1090 | 1060 | 1050 | 1035 | |
| 3 | Black cock .. | 1400 | 1420 | 1430 | 1440 | 1460 | 1405 | |
| 4 | Yellow cock .. | 1065 | 1015 | 870 | Died | | | No degeneration of nerves. |
| 5 | Black cock .. | 1510 | 1465 | 1400 | 1390 | 1360 | 1340 | |
| 6 | Black cock .. | 1475 | 1460 | 1435 | 1410 | 1410 | 1415 | |

EXPERIMENT No. 40.—Nos. 1—6 Untreated Padi. Nos. 7—12 Padi
heated at 120°C for two hours.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|------------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Yellow cock .. | 1140 | 1210 | 1175 | 1140 | 1135 | |
| 2 | Speckled cock .. | 1500 | 1500 | 1545 | 1400 | 1420 | |
| 3 | Red cock .. | 1600 | 1640 | 1660 | 1600 | 1625 | |
| 4 | Yellow cock .. | 1340 | 1340 | 1310 | 1310 | 1370 | |
| 5 | Red cock .. | 1505 | 1550 | 1575 | 1555 | 1550 | |
| 6 | Yellow cock .. | 1295 | 1340 | 1375 | 1340 | 1325 | |
| 7 | Black cock .. | 1540 | 1555 | 1535 | 1495 | 1375 | |
| 8 | White cock .. | 1715 | 1680 | 1630 | Polyneuritis | | |
| 9 | Speckled cock .. | 1540 | 1520 | 1490 | 1420 | Polyneuritis | |
| 10 | Yellow cock .. | 1710 | 1680 | 1715 | 1485 | Polyneuritis | |
| 11 | White cock .. | 1240 | 1200 | 1110 | Polyneuritis | | |
| 12 | Red cock .. | 1400 | 1360 | 1340 | 1240 | Polyneuritis | |

EXPERIMENT No. 41.—Nos. 1—4 Unpolished Rice.

Nos. 5—8 Unpolished Rice sterilised one hour.

Nos. 9—12 Unpolished Rice sterilised two hours.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Yellow cock .. | 1230 | 1100 | 1255 | 1210 | 1240 |
| 2 | Brown cock .. | 1075 | 1075 | 1140 | 1165 | 1155 |
| 3 | White cock .. | 1460 | 1490 | 1325 | 1485 | 1530 |
| 4 | Brown cock .. | 1355 | 1350 | 1205 | 1280 | 1305 |
| 5 | White cock .. | 1410 | 1305 | 1150 | 1005 | |
| 6 | Brown cock .. | 1250 | 1060 | 890 | 830 | |
| 7 | White cock .. | 1290 | 1300 | 1020 | Polyneuritis | |
| 8 | Brown cock .. | 1225 | 1125 | 950 | 855 | Polyneuritis |
| 9 | White cock .. | 1125 | 1065 | 945 | Polyneuritis | |
| 10 | Brown cock .. | 1170 | 1080 | 1000 | Polyneuritis | |
| 11 | Black cock .. | 1365 | 1365 | 1160 | 1045 | |
| 12 | Brown cock .. | 1045 | 975 | 870 | 840 | Polyneuritis |

EXPERIMENT No. 42.

Padi submerged in water and sterilised at 120°C.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Yellow cock .. | .. | 1240 | 1200 | 1220 | 1205 | 1275 |
| 2 | Brown cock .. | .. | 1155 | 1165 | 1170 | 1180 | 1170 |
| 3 | Black cock .. | .. | 1530 | 1520 | 1535 | 1420 | 1505 |
| 4 | Brown cock .. | .. | 1305 | 1280 | 1270 | 1295 | 1285 |
| 5 | White cock .. | .. | 1425 | 1395 | 1410 | 1450 | 1455 |
| 6 | Black cock .. | .. | 1340 | 1250 | 1200* | 1335 | 1320 |
| | | | | | | | 1295 |

EXPERIMENT No. 43.—Padi sterilised 120°C Dry heat.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week | 4th
week. | 5th
week. |
|-----|------------------|-----------------|--------------|--------------|-------------|--------------|--------------|
| 1 | Red cock .. | 1250 | 1230 | 1235 | 1250 | 1235 | 1220 |
| 2 | Speckled cock .. | 1560 | 1590 | 1560 | 1560 | 1530 | 1515 |
| 3 | Red cock .. | 1250 | 1210 | 1225 | 1235 | 1220 | 1220 |
| 4 | Grey cock .. | 1435 | 1370 | 1372 | 1375 | 1365 | 1355 |
| 5 | Black cock .. | 1775 | 1775 | 1880 | 1950 | 1975 | 1965 |
| 6 | Black cock .. | 1535 | 1455 | 1460 | 1500 | 1535 | 1555 |

EXPERIMENT No. 44.—Parboiled Rice cooked in open vessel.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | White cock .. | 1295 | 1200 | 1145 | 1180 | 1200 | 1235 |
| 2 | White cock .. | 1110 | 1035 | 1070 | 1080 | 1120 | 1140 |
| 3 | Grey cock .. | 1235 | 1190 | 1155 | 1140 | 1220 | 1170 |
| 4 | Black cock .. | 1075 | 1030 | 980 | 940 | 1055 | 980 |
| 5 | White cock .. | 1335 | 1510 | 1515 | 1510 | 1560 | 1695 |
| 6 | Black cock .. | 1135 | 1115 | 1015 | 1110 | 1110 | 1085 |

EXPERIMENT No. 45.—Parboiled Rice cooked by steam under pressure.

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week. | 5th week. |
|-----|----------------|-----------------|--------------|--------------|--------------|------------------------------|----------------|
| 1 | White cock .. | 1300 | 1185 | 1055 | 940 | 1000 | 855 |
| 2 | Black cock .. | 1030 | 930 | 845 | 760 | Polyn neuritis | |
| 3 | Red cock .. | 1070 | 1105 | 915 | 810 | 810 | 670 |
| 4 | Brown cock .. | 1220 | 1200 | 1165 | 1055 | 960 | Polyn neuritis |
| 5 | Yellow cock .. | 1230 | 1140 | 1110 | 970 | 950 | 890 |
| 6 | White cock .. | 1120 | 975 | 825 | 670 | Illness. Not Polyn neuritis. | |

was fed to a group of fowls. The cooking process involved exposure to steam under a pressure of two atmospheres at 120°C or thereabouts for two and a half hours. The fowls in this group rapidly lost weight and cases of polyneuritis developed among them.

These experiments showed that physical conditions other than temperature influenced the result, immersion in water for example in the case of padi seemed to negative entirely the destructive effect of an atmosphere of steam under the conditions which obtain in an autoclave. This series of experiments, has not been carried to a conclusion, but there is evidence that the destructive effect of high temperatures is complicated by the consideration of other physical conditions.

It was evident however that methods of analysis involving exposure of the materials to high temperatures would not enable us to determine the nature of the substances for which we sought and recourse was had to other methods.

Experiments to Isolate the Protective Substances contained in Rice Polishings.

From this point researches were carried out for the isolation of the substance or combination of substances in polishings which were responsible for this result.

For the purpose of testing the value of the various materials fowls weighing 1200 grms. or thereabouts were employed. Each, as in previous experiments, was confined in a separate cage. The fowls received rice twice daily at 10 a.m. and 3 p.m. and when receiving polishings or materials prepared from polishings the substance in question was given as an emulsion by means of a stomach-tube half an hour after the rice had been given. Every fowl was weighed once a week at 12 noon.

As the result of a series of observations it had been determined that fowls weighing from 1200 to 1400 grms. required about 60 grms. of unpolished rice daily and, if fed on 60 grms. of the polished rice used in these experiments they required in addition 5 grms. of sifted polishings for the maintenance of weight and health.

In a previous experiment where products derived from different lots of padi were employed, 3.5 grms. of the polishings were shown to be sufficient with the white rice then in use. In the present experiment all the products employed unpolished rice, polished rice, polishings, etc., were derived from the same lot of padi.

For purposes of comparison the following results of analyses are given:—

| | | | Protein. | Fats. | Carbo-
hydrates. | Ash. | Moisture. | Percent-
age of
P ₂ O ₅ . |
|-------------------------|-----|-----|----------|-------|---------------------|------|-----------|---|
| Polishings (sifted) ... | ... | ... | 13.7 | 14.16 | 52.77 | 7.54 | 11.83 | 4.1 |
| Unpolished rice ... | ... | ... | 9.0 | 1.65 | 75.52 | 1.68 | 12.75 | 0.56 |
| Polished rice ... | ... | ... | 8.6 | 0.22 | 76.23 | 0.6 | 14.35 | 0.26 |

When the composition of these articles is calculated on dried materials the differences are rendered more striking and accurate, and when in a similar manner the composition of a diet made up of 60 grms. of polished rice and 5 grms. of polishings is calculated it will be seen how closely such a diet approximates to one of unpolished rice.

CALCULATED ON DRIED MATERIALS.

| | | | Protein. | Fats. | Carbo-
hydrates. | Ash. | Percent-
age of
P_2O_5 |
|---|-----|-----|----------|-------|---------------------|------|--------------------------------|
| Polishings (sifted) | ... | ... | 15.5 | 16.0 | 60.0 | 8.5 | 4.65 |
| Unpolished rice | ... | ... | 10.3 | 1.89 | 86.58 | 1.23 | 0.64 |
| Polished rice | ... | ... | 10.0 | 0.25 | 89.05 | 0.7 | 0.3 |
| Ration 60 grams polished rice plus 5 grams.
polishings contains per cent | ... | ... | 10.4 | 1.5 | 86.8 | 1.3 | 0.64 |

Sifted polishings were invariably employed because polishings as received from the millers contain a considerable admixture of husk and broken rice.

Polishings when fresh are neutral in reaction but on keeping they become acid. This change does not impair their efficiency however and polishings which have been stored with ordinary care for months are quite as valuable as the fresh materials. The ordinary process of cooking does not impair the value of polishings. For these reasons it is considered that the essential substance or substances are not unstable.

Experiments to determine the value of Fat in Polishings.

Fat in the rice-grain is mostly confined to the subpericarpal layers. Unpolished rice is therefore richer in fat than polished rice and polishings are very rich in fat.

To determine the value of this fat a quantity of sifted polishings was packed in a percolator and percolated repeatedly with Petroleum Ether. In this way the amount of fat in the polishings was reduced from 14.16% to 0.6%. The fat-free polishings were dried by exposure to the sun until free from Petroleum Ether.

Twelve fowls were fed on polished rice and received in addition daily 4.5 grms. of fat-free polishings, being the approximate equivalent of 5 grms. of sifted polishings (Experiment No. 46). The fowls remained healthy and maintained their weight just as had been the case when fowls received polished rice and sifted polishings. The non-importance of fat was therefore decided and its exclusion from the number of possibilities was of the utmost value since the fat had hitherto complicated our experiments.

Experiments with the Substances Soluble in 0.3% Hydrochloric Acid.

Estimations of the percentage of Phosphorus Pentoxide in rices had consistently shown their value as indicators of the liability or otherwise of a given rice to produce polyneuritis. Thus the higher the percentage of Phosphorus Pentoxide contained in a rice the less liable was that rice to produce polyneuritis when fed to fowls.

The unpolished rice employed contained 0.56% Phosphorus Pentoxide and did not cause polyneuritis. The polished rice contained 0.26% Phosphorus Pentoxide and invariably caused polyneuritis, while washed rice containing 0.22% Phosphorus Pentoxide was more harmful than the unwashed polished rice. This suggested the probability that the essential substance was one containing Phosphorus.

Now it was known that a large percentage of the Phosphorus compounds present in rice polishings were soluble in 0.3% Hydrochloric Acid.

An experiment was therefore carried out to determine if when polishings were treated with 0.3% HCl, the physiologically active substances were removed.

Polishings in quantities of 180 grms. being the amount required by twelve fowls in three days, were mixed with 1000 cc. 0.3% Hydrochloric Acid, stirred during the day and

EXPERIMENT No. 46.

White Polished Rice washed + Polishings fat free.

| No. | Description. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | 5th week. |
|-----|---------------|--------------|-----------|-----------|-----------|-----------|-----------|
| 1 | Black hen .. | 1510 | 1525 | 1340 | 1360 | 1395 | 1360 |
| 2 | White cock .. | 1225 | 1230 | 1220 | 1215 | 1235 | 1255 |
| 3 | Red cock .. | 1700 | 1840 | 1900 | 1855 | 1895 | 1860 |
| 4 | Brown hen .. | 1215 | 1220 | 1305 | 1310 | 1340 | 1295 |
| 5 | Red cock .. | 1350 | 1515 | 1460 | 1430 | 1460 | 1450 |
| 6 | Black cock .. | 1465 | 1520 | 1500 | 1485 | 1520 | 1530 |
| 7 | Brown hen .. | 1445 | 1420 | 1290 | 1335 | 1330 | 1305 |
| 8 | White hen .. | 1685 | 1650 | 1750 | 1755 | 1745 | 1665 |
| 9 | Brown hen .. | 1225 | 1195 | 1195 | 1085 | 1070 | 1035 |
| 10 | Red cock .. | 1300 | 1350 | 1390 | 1380 | 1370 | 1380 |
| 11 | Black hen .. | 1265 | 1235 | 1220 | 1220 | 1230 | 1235 |
| 12 | White cock .. | 1410 | 1410 | 1390 | 1390 | 1380 | 1340 |

| | Protein. | Fat. | Carbohydrate. | Ash. | Moisture. | P ₂ O ₅ |
|----------------------------|----------|------|---------------|------|-----------|-------------------------------|
| White Polished Rice washed | 8.2 | .22 | 75.04 | 0.34 | 16.2 | .21 |
| Polishings Fat free | | | | | | .67 |

the following morning filtered through a Buchner's filter. 100 cc. of 0.3% Hydrochloric Acid were used to wash out the vessels. When fluid could no longer be extracted from the mass it was mixed with 600 cc. of 0.3% Hydrochloric Acid stirred during two hours and thereafter filtered as before.

The extracted polishings were mixed with distilled water, nearly neutralized with Sodium Carbonate, and the volume adjusted to 1080 cc. 30 cc. of this emulsion contained 5 grms. of polishings less the materials dissolved out by the acidulated water.

The combined filtrates obtained from 180 grms. of polishings were nearly neutralized with Sodium Carbonate and concentrated at a low temperature to a volume of 1080 cc. 30 cc. of this suspension contained the substances solved out by acidulated water from 5 grms. of polishings.

Twelve fowls were obtained and fed on washed polished rice, each receiving daily 30 cc. of the emulsion of extracted polishings (Experiment No. 47). Cases of polyneuritis occurred. This experiment was repeated (Experiment 48) with similar results.

Twelve fowls were fed on washed polished rice each receiving in addition 30 cc. of the suspension of dissolved substances (Experiment No. 49). Cases of polyneuritis did not occur.

When 100 grammes of polishings are extracted in the manner described 26 grammes of solids pass into solution. In this 26 per cent. therefore of the original polishings are contained the physiologically active substances.

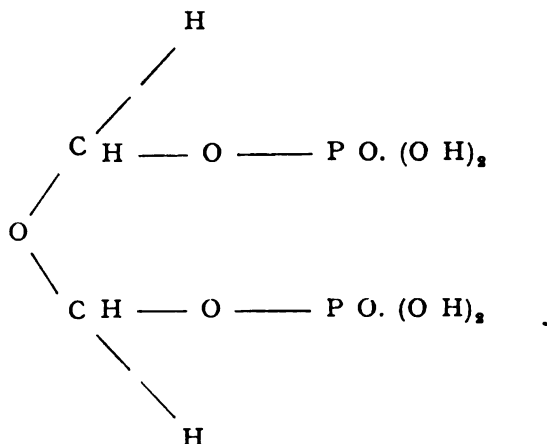
Experiments with Phytin.

It having thus been made clear that the substances of physiological importance in polishings were removed unchanged

by extracting with 0.3% HCl, the further research consisted in division of this fraction by various methods.

Of the substances contained in rice polishings which are soluble in 0.3% Hydrochloric Acid an important constituent is the phosphorus compound Phytin. Dr. Hans Aron has claimed that Phytin is of value in preventing the onset of polyneuritis in fowls fed on a white rice diet. The results of Dr. Arons experiments with this compound do not appear however to justify the claims that he has made for it.

Phytin was first isolated by Posternak from plant-seeds and the structural formula stated by him to represent phytic acid is



Phytin is stated to be the Calcium Magnesium salt of this acid and assuming the four hydroxyl groups to be replaced by one atom each of Calcium Magnesium, the salt would contain 8.05% of Magnesium and 13.4% of Calcium. These are lesser amounts than those recorded by Contardi who by actual analysis found 8.97% of Magnesium and 13.8% of Calcium.

The quantity of Phytin in rice was estimated as follows. A weighed quantity of rice was reduced to coarse powder shaken with 0.3% Hydrochloric Acid and then filtered. The residue

EXPERIMENT No. 47.—White Polished Rice + Polishings
freed from substances soluble in 0.3% HCl.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | 1235 | 1165 | 1115 | 1032 | |
| 2 | Brown hen .. | 980 | 960 | 885 | 850 | |
| 3 | Red cock .. | 1940 | 1770 | 1635 | 1465 | |
| 4 | Black hen .. | 1285 | 1230 | 1140 | 1050 | |
| 5 | Red cock .. | 1405 | 1200 | 1070 | 915 | Polyneuritis |
| 6 | Black cock .. | 1560 | 1460 | 1440 | 1285 | Polyneuritis |
| 7 | Black hen .. | 1520 | 1415 | 1340 | 1230 | Polyneuritis |
| 8 | Brown hen .. | 1350 | 1145 | 1090 | 1022 | Polyneuritis |
| 9 | Grey hen .. | 1375 | 1240 | 1135 | 1055 | Polyneuritis |
| 10 | White hen .. | 1510 | 1450 | 1310 | 1225 | |
| 11 | Red cock .. | 1705 | 1645 | 1515 | 1440 | Polyneuritis |
| 12 | Black hen .. | 1400 | 1235 | 1140 | 1050 | |

EXPERIMENT No. 48.

White Polished Rice washed + Polishings (0.3% HCl insoluble fraction)

| No. | Description. | Original wt. | 1st week. | 2nd week. | 3rd week. | 4th week. | — |
|-----|---------------|--------------|-----------|-----------|-----------|----------------|----------------------------------|
| 1 | Black hen .. | 1360 | 1325 | 1215 | 1140 | | |
| 2 | White cock .. | 1255 | 1150 | 1060 | | Polyn neuritis | |
| 3 | Red cock .. | 1860 | 1775 | 1645 | 1695 | Polyn neuritis | |
| 4 | Brown hen .. | 1295 | 1220 | 1210 | 1170 | | |
| 5 | Red cock .. | 1450 | 1335 | 1145 | Died | | No degeneration of nerve fibres. |
| 6 | Black cock .. | 1530 | 1520 | 1435 | 1480 | Polyn neuritis | |
| 7 | Brown hen .. | 1305 | 1270 | 1145 | 1080 | | |
| 8 | White hen .. | 1665 | 1605 | 1500 | 1390 | | |
| 9 | Brown hen .. | 1035 | 1040 | 985 | 920 | | |
| 10 | Red cock .. | 1380 | 1390 | 1300 | 1185 | Polyn neuritis | |
| 11 | Black hen .. | 1235 | 1215 | 1145 | 1080 | | |
| 12 | White cock .. | 1340 | 1335 | 1280 | | Polyn neuritis | |

EXPERIMENT No. 49.—White Polished Rice + Polishings (0.3% HCl. soluble fraction).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|------------------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Black hen .. | 940 | 1000 | 1010 | 1080 | 1125 | 1000 |
| 2 | Black and brown cock.. | 1440 | 1545 | 1615 | 1645 | 1665 | 1620 |
| 3 | Yellow cock .. | 1520 | 1600 | Died. | | | |
| 4 | Black hen .. | 1285 | 1390 | 1360 | 1300 | 1320 | 1305 |
| 5 | Brown hen .. | 1110 | 1195 | 1255 | 1240 | 1135 | 1160 |
| 6 | Yellow cock .. | 1680 | 1775 | 1840 | 1915 | 1895 | 1750 |
| 7 | Brown hen .. | 1185 | 1125 | 1055 | 1060 | 1035 | 1060 |
| 8 | Brown hen .. | 1425 | 1445 | 1515 | 1540 | 1485 | 1520 |
| 9 | Brown cock .. | 1305 | 1275 | 1390 | 1475 | 1480 | 1460 |
| 10 | Yellow hen .. | 1010 | 1160 | 1265 | 1230 | 1190 | 1200 |
| 11 | Yellow hen .. | 1170 | 1135 | 1155 | 1105 | 1145 | 1160 |
| 12 | Yellow hen .. | 1315 | 1290 | 1330 | 1230 | 1180 | 1160 |

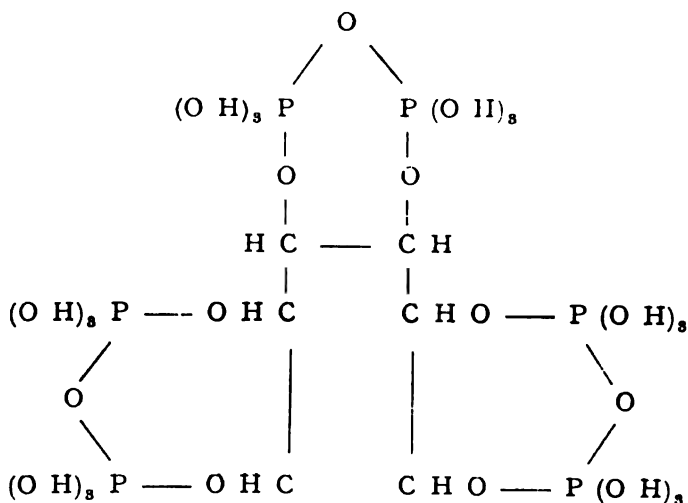
No degeneration of nerves.

was again shaken with acidulated water, this process being repeated until the filtrate ceased to give a precipitate with either Copper Acetate or Alcohol. To the combined filtrates excess of Copper Acetate was added. The mixture was allowed to stand overnight, thereafter the precipitate was collected, washed, dried, and weighed. From that weight, on the assumption that each molecule of the Copper phytinate contained two atoms of Copper, the amount of Phytin was calculated.

It is admitted that the process is not strictly speaking an accurate one but it was the best available and was satisfactory for purposes of comparison between various kinds of rice and their content of this highly phosphorized compound.

The unpolished rice in use was by this method shown to contain 1.07% of Phytin, the same rice after polishing yielded only a trace and the polished rice after washing in water contained none.

The structural formula for Phytic Acid as given by Neuberg is more complex than that of Posternak and has three times the molecular weight.



Phytin for experimental purposes was prepared by the following method. Sifted polishings were mixed with 0.3 per cent. Hydrochloric Acid in the proportion of 300 grms. of the former to 2000 cc. of the latter, the mixture was stirred throughout the day and on the following morning was filtered through a Buchner's filter. The clear yellowish filtrate was mixed with one and a half times its volume of 95 per cent. Alcohol which produced a white precipitate of Phytin; the mixture was allowed to stand for a few days. The precipitate was collected washed with strong alcohol to free it from acid and dried in a vacuum dessicator. A friable cake of Phytin was obtained readily reducible to a white powder, soluble in water, yielding an opalescent solution with an acid reaction and giving on addition of Sodium Carbonate a white flocculent precipitate.

As shown on page 74, 100 grammes of sifted polishings yield an average of 8.47 grammes Phytin. Unpolished rice loses 10% of its weight on polishing and on this basis the percentage of Phytin in unpolished rice would be not less than 0.847%. In our experiments to test the value of Phytin it was assumed that unpolished rice contained 1.07% of that substance and thereby if an error did exist it was in favour of the Phytin.

The Phytin prepared by us contained 34.8% of Phosphorus Pentoxide, whereas according to the formula of Posternak it should have contained 47.6% we proved the presence of Protein in the Phytin and we considered that our preparation contained 73% of the Calcium Magnesium salt of an acid having the formula given by Posternak.

A fowl consuming 60 grms. of unpolished rice daily would be receiving 0.64 grms. of Phytin. A fowl receiving the same amount of washed polished and therefore Phytin-free rice would require to have in addition that amount of Phytin daily in order to bring the value of this diet in respect of Phytin up to that of an unpolished rice diet.

Two experiments were carried out with this compound. In the first (No. 50) twelve fowls received washed polished rice and in addition Phytin, which was given in the following manner; 9 grms. of Phytin were dissolved in distilled water, the solution neutralized with Sodium Carbonate and the volume made up to 360 cc. Each fowl received 15 cc. of this suspension at 10.30 a.m. and 3.30 p.m. daily. All the fowls lost weight and cases of polyneuritis occurred just as if the fowls had received washed polished rice only.

In this experiment the fowls had the phosphorus pentoxide content of their diet augmented to 0.69% an amount in excess of that contained in a diet of unpolished rice but which on the assumption that our Phytin was only of 73% purity meant that they received daily only 0.54 grms. of Phytin in place of 0.64 gm.

In the next experiment (No. 51) twelve fowls received daily 1 gm. of Phytin which was prepared as an emulsion and intimately mixed with the washed polished rice but the results were the same.

Again assuming that our Phytin was only of 73% purity each fowl received daily 0.73 gm. of Phytin an amount sufficiently in excess of the calculated amount and certainly considerably in excess in respect of Phosphorus Pentoxide.

As shown subsequently the alcoholic filtrate from which the Phytin had been removed, freed from alcohol, was effective in protecting fowls on unpolished rice from the occurrence of polyneuritis. The importance of Phytin has therefore been disproved.

As the precipitate phytin had been shown to be ineffective, on theoretical grounds it was assumed that the whole of the active substances were contained in the filtrate.

The fraction remaining in solution after the precipitation of Phytin was next tested by the following procedure.

Polishings in quantities of 180 grms. were extracted with 0.3% Hydrochloric acid in the manner described on page 68 and the combined filtrates from each 180 grms. were mixed with one and a half times their volume of 95% alcohol. The precipitate was filtered off. A large number of weighings of this precipitate (Phytin) were made and it was found that an average of 8.47 grammes of Phytin were obtained from 100 grammes of sifted polishings.

The alcoholic filtrate was then nearly neutralised with Sodium Carbonate and evaporated at a low temperature until free from alcohol. The residue was diluted with distilled water to a volume of 1080 cc., 30 cc. of this suspension contained the almost Phytin-free soluble substances from 5 grms. of polishings.

Two experiments were carried out with this solution.

In the first (Experiment 52) six fowls were fed on washed polished rice, each receiving daily 30 cc. of this suspension. All remained healthy.

In the second (No. 53) twelve fowls were employed with a similar result.

That fraction of the substances originally soluble in 0.3% HCl, which still remains in solution on the addition of alcohol, *the acid soluble, alcohol soluble part*, have thus been shown to contain the whole of the substances physiologically active, and an attempt was made to further divide this fraction by alkalinising.

An experiment carried out with the precipitate, *the acid soluble, alcohol soluble, alkali precipitable part*, and another experiment with the filtrate, *the acid soluble, alcohol soluble, alkali soluble part*, had the unexpected result that the substances sought for were found to be no longer physiologically active in either fraction.

EXPERIMENT No. 50.—White Polished Rice + Polishings
(0.3% HCl soluble, Proof spirit insoluble, fraction).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd week. |
|-----|------------------|-----------------|--------------|--------------|---------------------|
| 1 | Brown hen .. | .. | 980 | 970 | 805 |
| 2 | Red cock .. | .. | 2000 | 1895 | 1700 Polyn neuritis |
| 3 | Red cock .. | .. | 1980 | 1920 | 1775 Polyn neuritis |
| 4 | Brownish cock .. | .. | 1395 | 1360 | 1315 Polyn neuritis |
| 5 | Brown hen .. | .. | 1790 | 1750 | 1670 1565 |
| 6 | Brown hen .. | .. | 1070 | 1050 | 980 865 |
| 7 | White hen .. | .. | 1235 | 1240 | 1135 1025 |
| 8 | Red hen .. | .. | 1205 | 1210 | 1170 1060 |
| 9 | Brown hen .. | .. | 1175 | 1000 | 885 770 |
| 10 | Grey hen .. | .. | 1150 | 1095 | 975 890 |
| 11 | Yellow hen .. | .. | 1150 | 1015 | 980 930 |
| 12 | Red hen .. | .. | 970 | 905 | 810 750 |

EXPERIMENT No. 51.—White Polished Rice + Polishings
(0.3% HCl soluble, Proof spirit insoluble, fraction.)

| No. | Description | Original
wt. | 1st
week. | 2nd
week. | 3rd week. |
|-----|-----------------------|-----------------|--------------|--------------|--------------|
| 1 | Red and Yellow cock | 1420 | 1370 | 1305 | 1150 |
| 2 | Red cock .. | 1310 | 1280 | 1207 | Polyneuritis |
| 3 | Red and black cock .. | 1180 | 1150 | 1140 | Polyneuritis |
| 4 | Yellow hen .. | 1040 | 990 | 910 | 815 |
| 5 | Black and red cock .. | 1340 | 1360 | 1290 | Polyneuritis |
| 6 | Yellow hen .. | 1240 | 1225 | 1160 | 1065 |
| 7 | Black hen .. | 940 | 995 | 970 | 960 |
| 8 | Yellow and black cock | 1285 | 1320 | 1157 | 1005 |
| 9 | Brown hen .. | 1380 | 1375 | 1335 | 1200 |
| 10 | Red and black cock .. | 1430 | 1420 | 1355 | 1215 |
| 11 | Yellow hen .. | 995 | 945 | 905 | 815 |
| 12 | Brown and black cock | 1050 | 1060 | 1005 | Polyneuritis |

EXPERIMENT No. 52.—White Polished Rice +
Polishings (0.3% HCl soluble, Proof spirit soluble, fraction.)

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|--------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | 2095 | 2140 | 2100 | 2060 | 2050 |
| 2 | Red cock .. | 1265 | 1280 | 1380 | 1275 | 1265 |
| 3 | Brown hen .. | 1550 | 1695 | 1655 | 1625 | 1660 |
| 4 | Brown hen .. | 900 | 850 | 905 | 915 | 935 |
| 5 | Brown hen .. | 1410 | 1430 | 1425 | 1445 | 1460 |
| 6 | Black hen .. | 1105 | 1160 | 1075 | 1070 | 1100 |

EXPERIMENT No. 53.—White Polished Rice + Polishings
(0.3% HCl soluble, Proof spirit soluble, fraction).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|------------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Yellow cock .. | 1248 | 1267 | 1310 | 1310 | 1215 |
| 2 | Speckled cock .. | 1330 | 1425 | 1450 | 1525 | 1560 |
| 3 | Red cock .. | 1590 | 1645 | 1650 | 1635 | 1605 |
| 4 | Yellow cock .. | 1350 | 1305 | 1325 | 1325 | 1350 |
| 5 | Red cock .. | 1557 | 1535 | 1595 | 1585 | 1565 |
| 6 | Black cock .. | 1174 | 1240 | 1345 | 1385 | 1390 |
| 7 | Red cock .. | 1510 | 1510 | 1590 | 1615 | 1635 |
| 8 | White cock .. | 1530 | 1675 | 1690 | 1760 | 1805 |
| 9 | Speckled cock .. | 1467 | 1480 | 1535 | 1600 | 1610 |
| 10 | Yellow cock .. | 1507 | 1575 | 1605 | 1730 | 1785 |
| 11 | White cock .. | 1170 | 1215 | 1280 | 1270 | 1250 |
| 12 | Red cock .. | 1430 | 1400 | 1400 | 1370 | 1390 |

Experiments with the Proof Spirit Filtrate.

Following on the demonstration that the effective substances sought for were contained in the filtrate and therefore soluble in alcohol of proof spirit strength, experiments were undertaken with a view to isolating and testing the value of the various substances contained in that solution.

The solution was found to contain substances giving the reactions characteristic of proteins. On increasing the alcoholic strength of this liquid a precipitate was produced and it was hoped that by sufficiently increasing the amount of alcohol the protein would be precipitated.

By experiments in which the proof spirit filtrate was nearly neutralised by means of Sodium Carbonate, freed from alcohol by evaporation at a low temperature, and the alcohol-free filtrate treated with varying quantities of 95% alcohol, it was found that seven volumes of this alcohol produced a precipitate which was not appreciably less than that produced when eight volumes of 95% alcohol were added. The addition of seven volumes of alcohol produced a mixture containing 85% of alcohol, with six volumes it contained 71%, and with eight volumes the mixture contained 84% of alcohol. The increase of alcoholic strength being therefore only 1% when eight volumes were employed in place of seven volumes and the amount of precipitate not being appreciably increased, it was decided to carry out an experiment in which the alcohol-free filtrate was treated with seven times its volume of 95% alcohol. The following procedure was adopted:

The proof spirit filtrate obtained from 30 grammes of polishings and measuring from 650—660 cc. was placed in a glass evaporating basin and partially neutralized. In all previous experiments the partial neutralization of this liquid had been effected by means of a solution of Carbonate of Soda of unknown

strength, but in order to secure consistency in this and all subsequent experiments a normal solution of Carbonate of Soda was employed. By experiment it was found that 13 cc. of this solution were required for neutralization of the proof spirit filtrate from 30 grammes of polishings; 10.5 cc. left the liquid slightly acid and it was decided that this amount should be added to each quantity of proof spirit filtrate evaporated. The partially neutralized liquid was evaporated at a temperature of 60°C until free from alcohol.

To the alcohol-free liquid seven times its volume of 95% alcohol were added, the mixture stirred, allowed to stand for two days and then filtered. The precipitate weighed on an average 0.7 gramme and consisted partly of Phytin; it was freed from alcohol by exposure to the air and suspended in 180 cc. of distilled water. This volume contained the substances in 30 grammes of polishings soluble in 0.3% HCl, soluble in proof spirit and insoluble in alcohol of 83% strength.

In Experiment No. 54 each of six fowls on white polished rice received daily in addition 30 cc. of this suspension, cases of polyneuritis occurred.

The 83% alcoholic filtrate was freed from alcohol by evaporation at a temperature of 60°C and to the alcohol-free residue distilled water was added to make the volume up to 180 cc. This volume contained the substances in 30 grammes of polishings soluble in 0.3% HCl, soluble in proof spirit and soluble in 83% alcohol.

In Experiment No. 55 each of six fowls on white polished rice received daily in addition 30 cc. of this solution and cases of polyneuritis did not occur.

It was observed that when the 83% alcoholic filtrate stood a few days a further slight precipitate occurred and in the next experiment the alcohol free liquid mixed with seven volumes of 95% alcohol was allowed to stand ten days and then filtered.

EXPERIMENT No. 54.—White Polished Rice + Polishings
(fraction soluble in 0.3% HCl, and in Proof spirit, and insoluble
in 83% Alcohol.)

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|--------------|-----------------|--------------|--------------|--------------|--------------|--------------|
| 1 | Spotted cock | .. | 1245 | 1300 | 1215 | 1100 | Polyneuritis |
| 2 | Black cock | .. | 1450 | 1490 | 1525 | 1540 | 1345 |
| 3 | Brown cock | .. | 1320 | 1350 | 1395 | 1405 | 1330 |
| 4 | White cock | .. | 1060 | 1145 | 1175 | 1105 | 1065 |
| 5 | Black cock | .. | 1290 | 1340 | 1375 | 1380 | 1325 |
| 6 | Brown cock | .. | 1510 | 1620 | 1665 | 1510 | 1315 |

EXPERIMENT No. 55.—White Polished Rice + Polishings
(fraction soluble in 0.3% HCl, soluble in Proof Spirit
and soluble in 83% Alcohol.)

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|----------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | .. | 1195 | 1245 | 1255 | 1235 |
| 2 | Red cock .. | .. | 1550 | 1525 | 1555 | 1545 |
| 3 | Yellow cock .. | .. | 1160 | 1160 | 1185 | 1130 |
| 4 | White cock .. | .. | 1120 | 1165 | 1200 | 1125 |
| 5 | Brown cock .. | .. | 1140 | 1185 | 1235 | 1185 |
| 6 | Red cock .. | .. | 1320 | 1315 | 1430 | 1440 |

The precipitate and filtrate were treated as in the preceding experiments.

In Experiment No. 56 each of six fowls on white polished rice received in addition daily their proportion of the 83% alcohol—insoluble substances and cases of polyneuritis occurred.

In Experiment No. 57 each of six fowls on white polished rice received in addition daily their proportion of the 83% alcohol—soluble substances and cases of polyneuritis did not occur.

It was thus proved that the protective substances were soluble in 83% alcohol and were not inactivated by contact with alcohol of that strength for a period of ten days.

In order that the strength of the alcoholic mixture might be considerably increased and the volume of the mixture kept within working limits absolute alcohol in place of 95% alcohol was employed in the next series of experiments. The procedure adopted was as follows:—

The proof spirit filtrate obtained from the 0.3% HCl solution prepared from 30 grammes of polishings and measuring from 650—660 cc. was placed in an evaporating basin, 10.5 cc. of normal solution of Carbonate of Soda was added and the liquid evaporated at a temperature of 60°C until the volume was reduced to 50 cc. To this was added 600 cc. of absolute alcohol, the mixture stirred, allowed to stand for two days and then filtered. By this method the mixture produced was one containing 91% of Ethyl Alcohol.

The precipitate weighed on the average one gramme and was therefore appreciably greater in amount than that obtained from a mixture containing 83% of alcohol. It was freed from alcohol by exposure to the air and suspended in 180 cc. of distilled water. This volume contained the substances in

30 grammes of polishings soluble in 0.3% HCl, soluble in proof spirit, and insoluble in 91% alcohol.

In Experiment No. 58 each of six fowls on white polished rice received in addition daily 30 cc. of this suspension and cases of polyneuritis occurred.

The 91% alcoholic filtrate was evaporated at a temperature of 60°C until free of alcohol, the residue dissolved in distilled water and the volume adjusted to 180 cc. This volume contained the substances in 30 grammes of polishings, soluble in 0.3% HCl, soluble in proof spirit, and soluble in 91% alcohol.

In Experiment No. 59 each of six fowls on white polished rice received in addition daily 30 cc. of this yellowish turbid fluid and cases of polyneuritis did not occur.

It was thus shown that the protective substances are soluble in 91% alcohol.

EXPERIMENT No. 56.—White Polished Rice + Polishings
(fraction soluble in 0.3% HCl, soluble in Proof spirit and
insoluble in 83% alcohol).

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|------------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | 1245 | 1180 | 1040 | Polyneuritis | |
| 2 | Brown cock .. | 1425 | 1390 | 1320 | 1260 | Polyneuritis |
| 3 | Speckled cock .. | 1405 | 1440 | 1420 | 1275 | Polyneuritis |
| 4 | White cock .. | 1265 | 1165 | 1140 | 1080 | Polyneuritis |
| 5 | Speckled cock .. | 1435 | 1350 | 1290 | 1175 | |
| | White cock .. | 1080 | 950 | 890 | 800 | |

**EXPERIMENT No. 57.—White Polished Rice + Polishings (fraction
soluble in 0.3% HCl, soluble in Proof spirit, and soluble
in 83% Alcohol).**

| No. | DESCRIPTION. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week. |
|-----|----------------|-----------------|--------------|--------------|--------------|-----------|
| 1 | White cock .. | 1325 | 1390 | 1465 | 1425 | 1410 |
| 2 | Black cock .. | 1465 | 1390 | 1480 | 1420 | 1400 |
| 3 | Black cock .. | 1470 | 1460 | 1570 | 1480 | 1475 |
| 4 | Yellow cock .. | 1250 | 1120 | 1140 | 1155 | 1190 |
| 5 | Black cock .. | 1350 | 1335 | 1395 | 1335 | 1330 |
| 6 | Red cock .. | 1275 | 1180 | 1240 | 1230 | 1215 |

EXPERIMENT No. 58.—White polished rice + Polishings
(fraction soluble in 0.3% HCl, soluble in Proof Spirit, and
insoluble in 91% Alcohol).

| No. | Description. | Original
wt | 1st
week. | 2nd
week. | 3rd
week. |
|-----|------------------|----------------|--------------|--------------|-------------------|
| 1 | Red cock .. | .. | 1320 | 1250 | 1140 Polyneuritis |
| 2 | Red cock .. | .. | 1450 | 1340 | 1335 Polyneuritis |
| 3 | Speckled cock .. | .. | 1300 | 1285 | 1275 1285 |
| 4 | Red cock .. | .. | 1420 | 1305 | 1185 Polyneuritis |
| 5 | Black cock .. | .. | 1495 | 1370 | 1430 1220 |
| 6 | Black cock .. | .. | 1360 | 1290 | 1285 1130 |

EXPERIMENT No. 59.—White Polished Rice + Polishings
(fraction soluble in 0.3% HCl, soluble in Proof spirit and soluble
in 91% alcohol).

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. |
|-----|------------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | Red cock .. | 1215 | 1285 | 1280 | 1285 | 1250 |
| 2 | Speckled cock .. | 1540 | 1600 | 1625 | 1630 | 1560 |
| 3 | Red cock .. | 1115 | 1190 | 1240 | 1270 | 1250 |
| 4 | Grey cock .. | 1250 | 1280 | 1360 | 1455 | 1435 |
| 5 | Black cock .. | 1550 | 1525 | 1675 | 1750 | 1775 |
| 6 | Black cock .. | 1415 | 1420 | 1570 | 1560 | 1535 |

Experiments with 91% Alcohol Filtrate.

Experiments were undertaken with a view to the isolation and testing of the various substances contained in the 91% alcoholic filtrate.

This filtrate was a clear yellowish liquid. In view of the fact that the protective substance or substances are destroyed by exposure to steam under pressure, it was considered probable that the activity of the liquid was not due to the presence of salts, but rather to the presence of some organic compound or compounds.

When the filtrate is freed from alcohol and the residue mixed with distilled water a yellowish turbid liquid is obtained. This liquid on saturation with Ammonium Sulphate yields a reddish brown precipitate which gives the usual protein reactions. Half saturation with Ammonium Sulphate produces a slight precipitate. Saturation with Sodium Chloride produces a precipitate and on filtration the filtrate saturated with Ammonium Sulphate gives a further precipitate.

As many vegetable globulins are not precipitated until their solutions are nearly saturated with Ammonium Sulphate it cannot be inferred that the alcoholic filtrate contains both a globulin and albumin.

Unpolished rice was tested and found to contain alcohol soluble proteins while polished rice similarly tested was found to contain none. It would appear therefore that Rosenheim and Kajuirra were in error in stating that rice did not contain alcohol-soluble proteins. It seems probable that they did not examine unhusked rice (padi) and unpolished rice not being an article of commerce would of course not be available.

The alcoholic filtrate freed from alcohol and the residue solved in distilled water was tested with Fehling's Solution and gave a precipitate of Cuprous Oxide. The solution was heated with Phenylhydrazine Hydrochlorate and Sodium Acetate; crystals of Phenyl-glucosazone were obtained. Another portion of the liquid was saturated with Ammonium Sulphate and filtered, the filtrate was heated with Phenylhydrazine Hydrochlorate and Sodium Acetate and crystals of Phenyl-glucosazone were obtained.

The 91% alcoholic filtrate evaporated to dryness and the residue dried in a dessicator was found to contain, after making allowance for the Sodium Chloride formed on partial neutralization with normal solution of Sodium Carbonate, total solids amounting to 14.2 % of the sifted polishings. These solids form a brown, sticky, hygroscopic residue.

Gravimetrically it was determined that 2.9 grammes of glucose, and by the Kjeldahl process that 2.08 grammes of protein are contained in this 14.2 grammes of total solids. The remaining 9.22 grammes was assumed to be salts.

It was sought to isolate and test the value of the alcohol soluble proteins. For this purpose dialysis was tried but the conditions here are unsuitable for dialysis, as even with the addition of Thymol, a mass of moulds and bacteria develop in a day or two both in the dialyser and the water. Unfortunately we had not a porcelain filter suitable for attachment to the water supply and eventually dialysis for the purpose of separating the alcohol-soluble proteins had to be abandoned.

It was decided to employ Ammonium Sulphate for the separation of these bodies and the following procedure was adopted:—

The 91% alcoholic filtrate, representing the quantity obtained from 30 grammes of polishings, was evaporated at 60°C till free from alcohol; the residue was dissolved in distilled water and

the volume adjusted to 100 cc. To this solution was added 72 grammes of Ammonium Sulphate being the amount found by experiment to be necessary for saturation. The mixture was agitated until the salt was entirely dissolved, then filtered.

The reddish brown precipitate obtained was solved in distilled water and the volume adjusted to 180 cc. This volume contained the substances in 30 grammes of polishings, soluble in 0.3% HCl, soluble in proof spirit, soluble in 91% alcohol and precipitated by saturation of the latter solution freed from alcohol with Ammonium Sulphate. The yellowish fluid obtained was a solution of the alcohol-soluble proteins in a dilute solution of Ammonium Sulphate. The fact that the proteins were soluble in this fluid suggests that they were not denatured.

In experiment No. 60 each of six fowls on white polished rice received in addition daily 30 cc. of this solution. It was calculated experimentally that each fowl received daily not more than 0.5 gramme of Ammonium Sulphate. All the fowls lost weight and one case of polyneuritis showing marked degenerative nerve-changes occurred in the fourth week.

The filtrate, obtained after saturation of the protein containing solution, was useless for feeding experiments because it was saturated with Ammonium Sulphate. This filtrate after standing a few days showed a deposit of crystals which on examination were found to be Magnesium Sulphate.

This filtrate freed from proteins and assuming that it contained only sugar and salts ought to be completely dialysable.

It was thought probable that this process might more readily be carried out on the protein-free fluid; but even in the case of this fluid, after two days dialysis in running water, the outside of the parchment paper was covered with a slime of micro-organisms. The addition of Thymol to the contents of the dialyser prevented growths occurring inside the dialyser but Thymol in the water did not prevent the growth on the outside.

Dialysis was only possible by the following procedure. The protein-free filtrate was placed in the dialyser along with a piece of Thymol; after two days dialysis in running water, the fluid was heated on a water bath, the parchment paper renewed and the fluid dialysed for another two days. It was heated again, the paper renewed and a further two days dialysis carried out. It was then heated and dialysed into distilled water changed daily for six days. The fluid remaining after dialysis for twelve days was evaporated to dryness and a slight residue of a yellowish colour was obtained.

The residue weighed 0.02 gramme, it was soluble in water, and partly organic. It amounted to not more than 0.02% of the solids contained in the fluid dialysed.

It was observed that the fowls fed on white rice *plus* the alcohol-soluble proteins lost weight and it was considered possible that part of these substances might have been lost in the course of various manipulations to which the fluid had been subjected. On account of the large quantities of absolute alcohol required it was not possible to prepare the alcohol soluble proteins in such quantities as to enable us to give each fowl daily the amount obtained from 10 grammes of polishings. It appeared simpler to employ the original proof spirit filtrate. It may here be remarked that the active substances are unchanged by contact for months with proof spirit. In this experiment the procedure adopted was as follows:—

The proof spirit filtrate in quantities representing the materials from 30 grammes of polishings dissolved out by 0.3% HCl, soluble in proof spirit and measuring usually from 650—660 cc. was placed in glass evaporating basins. To each lot 10.5 cc. of normal solution of Carbonate of Soda was added and evaporation carried out at 60°C.

The alcohol free fluid obtained from each lot was made up to 100 cc. with distilled water saturated with 72 grammes of Ammonium Sulphate and filtered.

The precipitate was dissolved in distilled water and the volume adjusted to 180 cc. This contained the substances in 30 grammes of polishings, dissolved out by 0.3% HCl, soluble in proof spirit, and precipitated by Ammonium Sulphate.

In Experiment No. 61 each of six fowls on white polished rice received in addition daily 30 cc. of this solution. At the end of one week all but one had lost weight and it was decided to give them an additional 30 cc. daily, the second dose being given half an hour after they had received the afternoon meal. Even with this increased amount the fowls continued to lose weight and at the close of the fourth week one case of what appeared to be polyneuritis occurred but examination of the nerves failed to reveal the presence of degenerative changes.

The experiment was continued for 31 days and on the thirtieth day another similar case occurred but again there were no degenerative changes in the nerves.

It must therefore be concluded that the alcohol-soluble proteins are not by themselves sufficient to protect fowls fed on white rice from the occurrence of polyneuritis. This conclusion is based on the assumption that the alcohol-soluble proteins were unchanged by the treatment to which they had been subjected.

In previous experiments it has been shown that the protective substances are extracted from parboiled rice by the use of hot 94% alcohol. It was thought possible that, as polishings are in a much finer state of sub-division, agitation with 95% alcohol might suffice to extract the protective substances and not the protein but even by this process both protein and glucose were extracted; a similar result was obtained with absolute alcohol. Experiments are now being undertaken to determine if the protective substance or substances can be separated from the Ammonium Sulphate filtrate or from polishings by the use of other solvents, as for example Ethyl acetate.

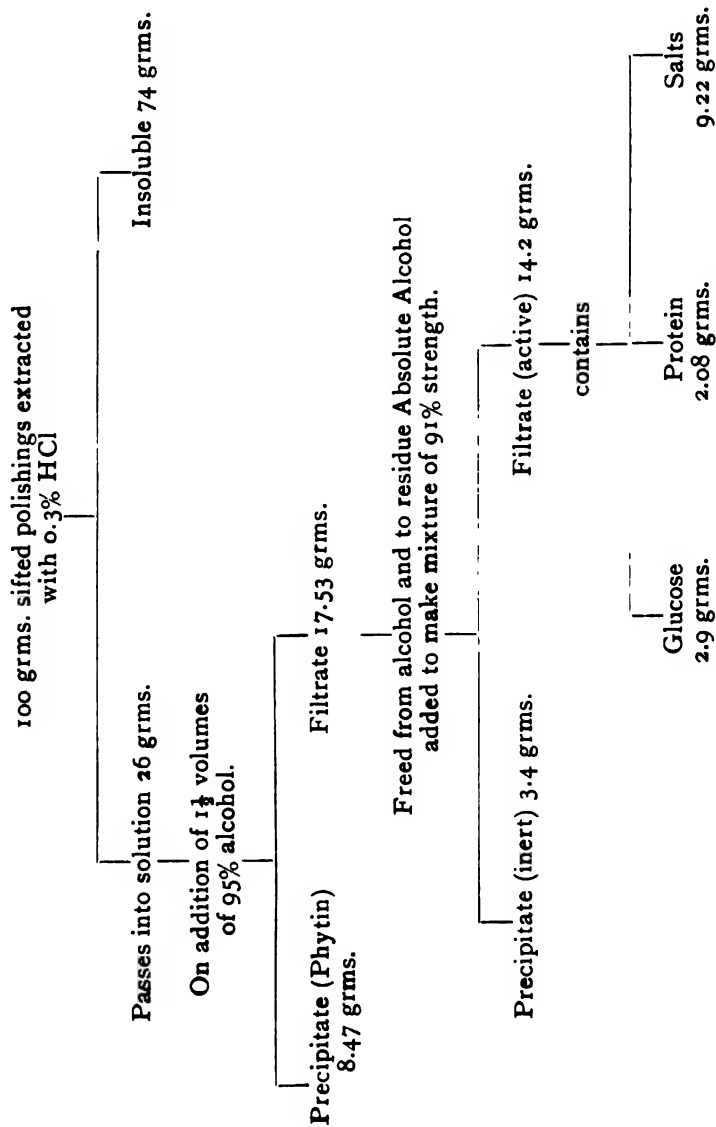
It may be that the 91% alcoholic filtrate contains substances other than Proteins, glucose, and salts, but until such time as the various constituents of that filtrate have been isolated, tested and identified, the biological reaction remains the only method by which the presence of the protective substances can be detected.

EXPERIMENT No. 60.—White Polished Rice + Ammonium
Sulph. precipitate from 91% alcohol filtrate.

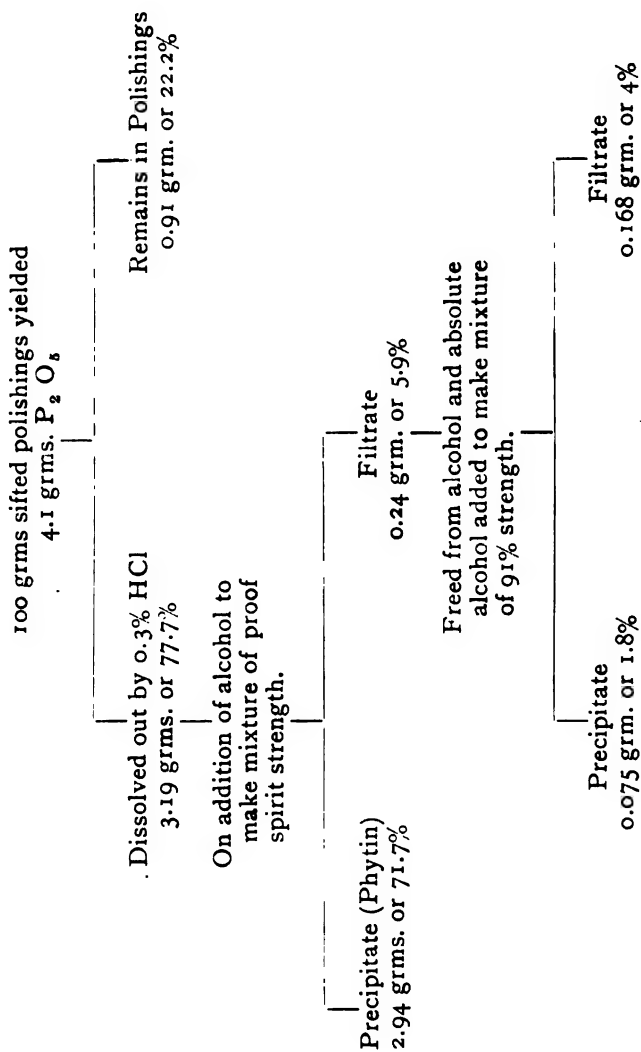
| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th week. |
|-----|---------------|-----------------|--------------|--------------|--------------|--------------|
| 1 | White cock .. | 1185 | 1075 | 955 | 950 | 910 |
| 2 | Brown cock .. | 1100 | 985 | 955 | 960 | 955 |
| 3 | Brown cock .. | 1370 | 1330 | 1340 | 1285 | 1245 |
| 4 | White cock .. | 1115 | 1105 | 1035 | 1025 | 1000 |
| 5 | Black cock .. | 1180 | 1145 | 1120 | 1065 | 1025 |
| 6 | Brown cock .. | 1165 | 1040 | 1005 | 915 | Polyneuritis |

EXPERIMENT No. 61.—White Polished Rice + Ammon. Sulph.
precipitate from Proof Spirit Filtrate.

| No. | Description. | Original
wt. | 1st
week. | 2nd
week. | 3rd
week. | 4th
week. | 5th
week. |
|-----|---------------|-----------------|--------------|--------------|--------------|---------------|--------------|
| 1 | Red cock .. | 1070 | 1040 | 1020 | 930 | 840 | |
| 2 | White cock .. | 1020 | 1030 | 1015 | 965 | Polynneuritis | |
| 3 | Brown cock .. | 1135 | 1090 | 980 | 895 | 810 | |
| 4 | Brown cock .. | 1270 | 1195 | 1080 | 970 | 785 | |
| 5 | Black cock .. | 1455 | 1415 | 1330 | 1230 | 1200 | |
| 6 | Brown cock .. | 1115 | 1025 | 1015 | 910 | 800 | |



NOTE.—These figures are based on an average of all observations and include many made since the publication of a paper in the *Lancet* No. 4555 of 17th December, 1910. They differ to a slight extent from those recorded in that paper.





| Effect on
fowls;
amount in
estimated
by the
occurrence
of
polynouritis. | Estimated
amount in
grms. of
fat in diet. | Estimated
amount in
grms. of
P ₂ O ₅ in
diet. |
|--|--|---|
| Unpolished rice..... | 0.99 | 0.336 |
| Polished rice plus polishings..... | 0.83 | 0.357 |
| Polished rice..... | 0.13 | 0.156 |
| Washed polished rice..... | 0.13 | 0.126 |
| Washed polished rice plus fat-free polishings..... | 0.16 | 0.326 |
| Washed polished rice plus polishings..... | 0.83 | 0.331 |
| Polishings extracted with 0.3% HCl. | | |
| Washed polished rice plus extracted polishings..... | 0.83 | 0.165 |
| Washed polished rice plus.....extract..... | 0.13 | 0.281 |
| Extract mixed with one and a half
times its volume of 95% Alcohol. | | |
| Washed polished rice plus.....precipitate..... | 0.13 | 0.27 |
| Washed polished rice plus.....filtrate..... | 0.13 | 0.138 |
| Filtrate freed from alcohol and
absolute alcohol added to residue
to make mixture of 91% strength. | | |
| Washed polished rice plus.....precipitate..... | 0.13 | 0.129 |
| Washed polished rice plus.....filtrate | 0.13 | 0.133 |



Conclusions.

1. The occurrence of Beri-beri in the Malay Peninsula has an intimate relationship with the consumption of a diet of which white polished rice forms the staple. Those who consume unpolished rice or slightly polished (native or Malay, or parboiled) rice do not suffer from the disease.

2. Fowls fed on white polished rice known to have been associated with outbreaks of human beri-beri develop a form of polyneuritis clearly analogous to Beri-beri in its clinical manifestations and pathological effects. Other white polished rices produce a similar result. Fowls fed on unpolished rice remain healthy.

These animals may therefore be employed to study the mode of operation by which a diet of white polished rice results in Beri-beri in man.

3. The estimation in terms of phosphorus pentoxide of the total phosphorus content of a given rice may be used as an indicator of the extent to which such a rice has been milled or polished and therefore of its Beri-beri producing power when forming the staple of a diet in man.

4. The harmful influence of white polished rice is not due to the existence in it of a poison developed after milling. White polished rice makes default in respect of some substance of high physiological importance essential for the maintenance of health.

5. Fowls fed on white polished rice constantly develop polyneuritis in a period of three to four weeks.

6. If the meal or polishings removed from such white rice in the process of milling be added to a diet of white polished rice, fowls remain healthy.

Substances essential for the maintenance of health are therefore contained in polishings.

7. Unpolished rice which has been submitted to sterilization in the autoclave at a temperature 120°C for two hours will cause polyneuritis when fed to fowls. The protective substances are destroyed under these conditions.

Methods of analysis involving exposure to high temperatures are therefore unsuitable for determining the nature of the protective substances.

8. The fats contained in the peripheral layers of the grain are of no value in protecting against polyneuritis.

9. The protective substances are soluble in 0.3% Hydrochloric Acid.

Phytin which comprises 32.5% of the substances so soluble is without value as a protective.

10. The substances are not precipitated from solution in 0.3% Hydrochloric Acid on the addition of 95% alcohol in such quantity as to make the resulting mixture of proof spirit strength.

They are soluble in proof spirit containing approximately 0.12% Hydrochloric Acid.

11. The protective substances are soluble in a slightly acidulated solution containing 91% of alcohol and, exclusive of glucose, amount to not more than 11.3% by weight of rice polishings and not more than 1.13% of the original unpolished rice grain. In this fraction are included prolamine (alcohol soluble protein) and compounds of calcium, magnesium and phosphorus.

These researches, which comprise an unbroken sequence of experiments beginning with rices associated with outbreaks of human beri-beri, demonstrate that rice is rendered harmful by the milling and polishing process to which it is subjected in

the preparation of white polished rice. In this process there is removed from the grain some substance of high physiological importance in the metabolism, the absence of which results in the production of polyneuritis in fowls and of beri-beri in man when a diet is consumed of which white polished rice is the staple. Whether these substances act by rendering other elements in the diet available for nutrition or whether they are themselves the nutritive material necessary for nerve tissues can in our present state of knowledge only be matter for conjecture. These substances, small in amount as compared with the total of the diet, have been determined within certain narrow limits but their exact chemical nature is still unknown.

There is no evidence that white rice contains a poison generated after decortication by the action of moulds or other organisms.

As measures for the prevention of beri-beri in this country it is recommended that the use of unpolished or under-milled rice be encouraged among those classes of the community in which the disease occurs. The polishing process if carried out at all should not extend beyond the removal of the outer skin or pericarp. The parboiling of rice before milling, as recommended by Dr. Braddon, serves the important purpose of so hardening the outer layers of the grain that their removal is less easy and over-milling is less likely to occur. The cooking of rice by steam under pressure should be prohibited. As an indicator of the extent to which rice has been milled we recommend to chemists the use of the phosphorus pentoxide standard. In the examination of a large number of rices, none were found associated with human beri-beri or polyneuritis in fowls which yielded a phosphorus pentoxide content of 0.4% or over, as estimated on the undried material. The amount of moisture varied only slightly and none of the rices were faced.

REFERENCES.

- (1). Wernich.—“Geographisch-medicinische Studien,” Berlin, 1878.
- (2). Van Leent.—“*Geneesk Tijdschr, voor Nederl-Indie*,” 1880.
- (3). Takaki.—“Prevention of Kakke,” in Japanese Navy, *Sei-i-Kwai*, 1885, 1886, 1887.
- (4). Idem.—“The Health of the Japanese Navy and Army,” *Lancet*, 1906, Nos. 1369, 1451, 1520.
- (5). Durham, H. E.—“Notes on Beri-beri in the Malay Peninsula and on Christmas Island,” *Journal of Hygiene*, 1904, Vol. IV, No. 1.
- (6). Braddon, W. L.—“The Etiology of Beri-beri,” *F. M. S. Medical Archives*, 1901.
“The Cause and Prevention of Beri-beri,” London, 1907.
- (7). Fletcher, W.—“Rice and Beri-beri,” *Lancet*, 1907, Vol. 1, p. 1776; “Rice and Beri-beri,” *Journal of Tropical Medicine and Hygiene*, 1909, No. 12, p. 127.
- (8). Fraser, H. and Stanton, A. T.—“An Inquiry Concerning the Etiology of Beri-beri,” *Lancet*, 1909, No. 4459, p. 451.
“An Inquiry Concerning the Etiology of Beri-beri,” Studies from the Institute for Medical Research, 1909, No. 10.
- (9). Schaumann, H.—“Die Aetiologie der Beri-beri unter Berücksichtigung des gesammten Phosphorstoffwechsels,” Leipzig, 1910.
- (10). Hooper, D.—“The Composition of Indian Rice,” *The Agricultural Ledger*, 1909, No. 5, Calcutta.

(11). Watt.—“The Commercial Products of India,” London, 1908.

(12). Eijkman, C.—“Eine Beri-beri-ähnliche Krankheit der Hühner,” *Vichows Archiv*, 1897.

“Polyneuritis bei hoenders, nieuwe tot de aetiologie der Ziekte,” *Geneesk. Tijdschr. v. Nederl. Indie*, 1896.

(13). Grijns, G.—“Over Polyneuritis gallinarum I, II, III,” *Geneesk. Tijdschr. voor Nederl. Indie*, 1901, 1909, 1910.

(14). Hulshoff Pol, D. J.—“Beri-beri. Voorkoming en genezing door toediening van Katjang hidjoe,” Amsterdam, 1904.

“Beri-beri en Katjang-hidjoe,” *Geneesk. Tijdschr. voor Nederl. Indie*, 1906.

“X—Zuur, het tegen Beri-beri werkzame bestanddeel uit de Katjang hidjoe,” *Geneesk. Tijdschr. voor Nederl. Indie*, 1907.

(15). Kiewiet de Jonge, G. W.—“Onderzoekingen over Beri-beri,” *Geneesk. Tijdschr. voor Nederl. Indie*, 1909.

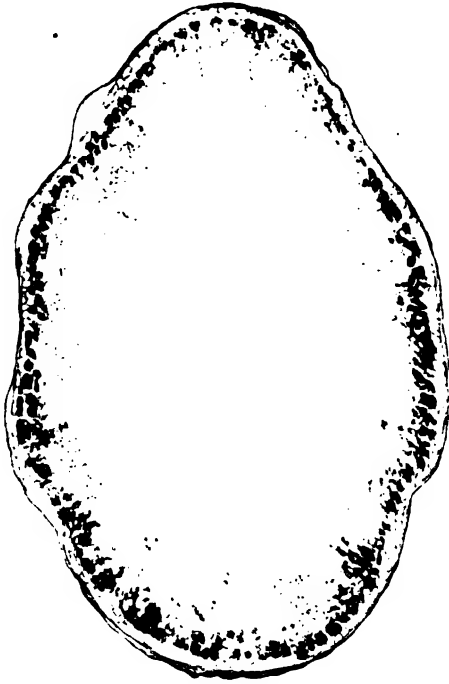
(16). Wright, H.—“Studies from the Institute for Medical Research,” Vol. II, No. 1, 1902.

(17). Daniels, C. W.—“Studies from the Institute for Medical Research,” Vol. IV, Pt. 1, 1906.

(18). Holst, A. and Fröhlich, T.—“Experimental Studies relating to Ship Beri-beri and Scurvy,” *Journal of Hygiene*, 1907, Vol. VII, No. 5.

(16). Aron, H.—“Phosphorus Starvation with special reference to Beri-beri,” *Philippine Journal of Science*, 1910, Vol. V, No. 1.

PLATE 1



A. J. E. Terai 1st

Bale & Danesong, 1st 1st

TRANSVERSE SECTION OF PADI (HUSK REMOVED)

PLATE II



A J E Terra del

Bale & Dickinson, ^{2d} lith

TRANSVERSE SECTION WHITE (POLISHED) RICE

24

RAI

PLATE III



A. J. E. Terzi, Jr.

Bale & Davidson I.M. 1941.

TRANSVERSE SECTION PARBOILED (UNPOLISHED) RICE



STUDIES
FROM
THE INSTITUTE FOR MEDICAL RESEARCH,
FEDERATED MALAY STATES.

- No. 1. Vol. 1.—No. 1. The Malarial Fevers of British Malaya by
HAMILTON WRIGHT, M.D. (McGILL).
- No. 2. Vol. 2.—No. 1. An Inquiry into the Etiology and Pathology
of Beri-Beri by HAMILTON WRIGHT, M.D. (McGILL).
- No. 3. Vol. 2.—No. 2. On the Classification and Pathology of Beri-
Beri by HAMILTON WRIGHT, M.D. (McGILL).
- No. 4. Vol. 3.—Part 1. The Diseases of British Malaya by C. W.
DANIELS, M.B. CAMB.
- No. 5. Vol. 3.—Part 2. Water Supplies (Preliminary Observations)
by C. W. DANIELS, M.B. CAMB.
- No. 6. Vol. 3.—Part 3. Breeding Grounds of Culicidæ by C. W.
DANIELS, M.B. CAMB. The Culicidæ of Malaya by G. F. LEICESTER,
M.B., C.M. EDIN.
- No. 7. Vol. 3.—Part 4. The Outbreaks of Rinderpest in Selangor,
1903 and 1904, by C. W. DANIELS, M.B. CAMB.
- No. 8. Vol. 4.—Observations on Beri-Beri by C. W. DANIELS, M.B. CAMB.
- No. 9. Surra in the Federated Malay States by HENRY FRASER, M.D.
(ABER.) and S. L. SYMONDS, V.S. (MELB.).
- No. 10. An Inquiry concerning the Etiology of Beri-Beri by HENRY
FRASER, M.D. (ABER.) and A. T. STANTON, M.D. (TOR).
- No. 11. The Etiology of Beri-Beri by HENRY FRASER, M.D. (ABER.) and
A. T. STANTON, M.D. (TOR).
- No. 12. The Etiology of Beri-Beri by HENRY FRASER, M.D. (ABER.) and
A. T. STANTON, M.D. (TOR).

TO

202 Main Library

HOME USE

Renewals and Recharges may be made 4 days prior to the due date.

Books may be Renewed by calling 642-3405

DUE AS STAMPED BELOW

JUL 24 1996

JAN 09 2000

UNIVERSITY OF CALIFORNIA, BERKELEY

BERKELEY, CA 94720

